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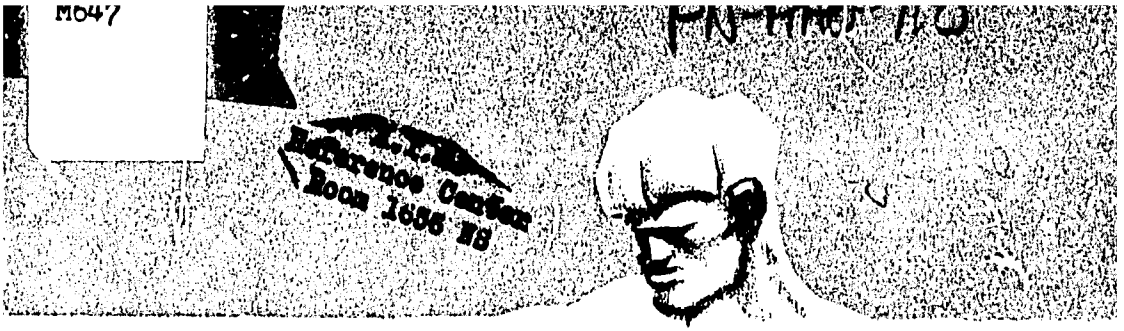

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WATER
AND MAN'S HEALTH

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WATER AND MAN'S HEALTH

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The publication of this monograph is a part of the world-wide program of the Agency for International Development and the World Health Organization for the improvement of public water supplies. Other publications including original articles, reprints, books and a wide variety of specifically developed reports are available in the English language. Some items have been translated into foreign languages. Readers seeking further information, materials, or technical assistance related to water supply development are invited to communicate with the representative of the U.S. Agency for International Development in the capital of their country or the Community Water Supply Branch, Health Service, Office of Human Resources and Social Development, Agency for International Development, Washington, D.C. 20523

FOREWORD

Developing community water supplies was initiated in many countries in 1959. One of the stimuli to the new undertakings was the resolution by the World Health Organization in 1958 at the Minneapolis, Minnesota, General Assembly meeting to increase emphasis on world-wide community water supply efforts. Preceding this resolution, in 1956, the Inter-American Committee of Presidential Representatives had endorsed and highlighted the importance of public water supply programs for the Western Hemisphere.

Supporting these various affirmations of need and feasibility is the 16 years of experience in such activities by the several predecessor agencies to the Agency for International Development. This experience has demonstrated the technical, administrative and economic soundness of water supply development for small communities as well as the problems involved.

Much is known about the chemical and biological relationships of water to human physiology but the general place of water in the ecology of man needs better focus. The fact that the world-wide development of water resources is of ever-increasing importance to the social and economic development of millions of people makes it important to have as precise knowledge of the water-man-health relationship as is possible.

Drawing upon his long experience in that aspect of public health endeavor which pertains especially to the environment, Mr. Miller has evolved an approach to a consideration of this relationship which should result in a better understanding of it. We take pride in the fact that the Agency for International Development, through its Community Water Supply Branch, has been able to make this addition to the general literature in the hope that it will serve to the benefit of all humanity.

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INTRODUCTION

> "Whoever wishes to investigate medicine properly should consider the seasons of the year, the winds and the waters in relation to health and disease." [1]

Water is a physiological necessity to man; it is also essential to agricultural and industrial development and growth. From time immemorial, man has accepted water wherever he found it and used it to maintain life and for other advantageous purposes. Many people in the world today still pursue this course, either by choice or through necessity. But all men were not forever satisfied with mere acceptance of available water; some conceived the idea that certain waters could be controlled and made more responsive to their needs. In ancient times, control actions took two forms: one, to protect and enhance quality, and another, to improve accessibility and availability.

The beneficial alteration of the quality of water may antedate 2000 B.C.[1] However, in that year "Ousruta Sanghita"—a collection of medical lore in Sanskrit—included a statement, according to Place,[2] that "It is good to keep water in copper vessels, to expose it to sunlight, and filter through charcoal." Other ancient records, including the Bible, refer to water storing, clarifying, filtering, and distilling to make it more palatable and less objectionable for hygienic use. From these days until the present time, man has continued his efforts to improve the palatability and potability of water.

History indicates that the collection of water to make it more easily procurable was not initiated until centuries after attention had been paid to its potability. It is said that when Caesar began taking possession of Alexandria in 47 B.C.,[1] he found the city underlaid with aqueducts designed to bring water from the Nile to cisterns in which it became clarified by sedimentation. In 97 A.D., Sextus Julius Frontinus was curator aquarium (water commissioner) of Rome and, among other works, he wrote a treatise on public water supply, De Aquis Urbis Romae Libri II (Two Books on the Water Supply of Rome). As civilization advanced, the need for pure water and the works to bring it to urban groups increased. Today the immensity of some water collection and purification works is astonishing.

As man increased in numbers, migrated to other land areas and clustered together for greater physical safety, he contributed more and more to the

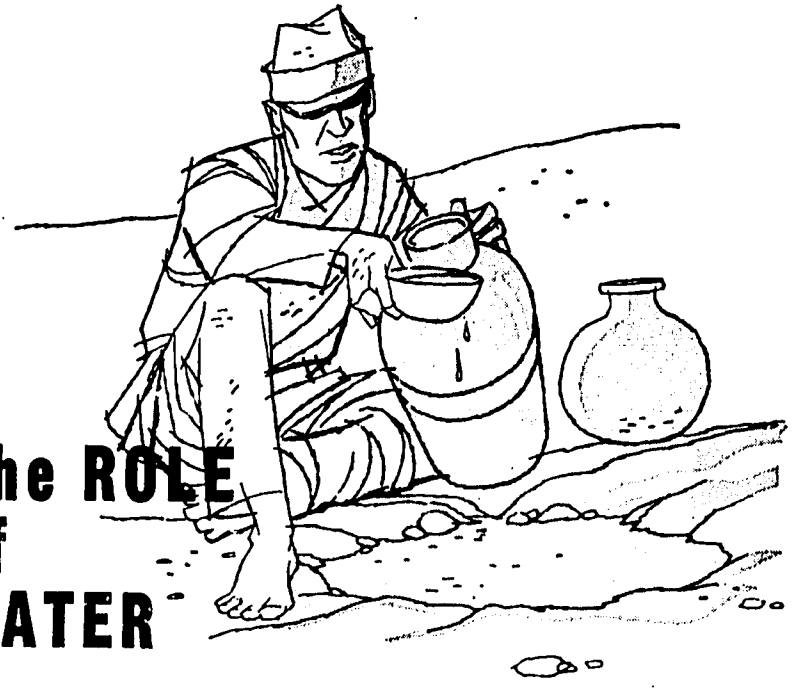
degradation of the water available to him. Diseases and bodily afflictions were not then so well identified and catalogued but people were just as vulnerable to them as they are today. With the improvement of the medical and biological sciences, recognition of the part played by water in the initiation and transmission of diseases and other bodily disturbances became more and more obvious until today there is a frightening array of agents which can exist in water and affect the susceptible person if and when the opportunity is presented.

This monograph identifies these agents and discusses ways to combat the bodily disturbances which they may cause.

REFERENCES

1. Hippocrates (460-354 B.C.) as quoted by Baker, M. N. The Quest for Pure Water. American Water Works Association, New York (1949).

2. Place, F. E. Water Cleansing by Copper. J. Prev. Med. (London), 13:379 (1905).



The ROLE of WATER

Henderson[3] states that "water is ingested in greater amounts than all other substances combined, and it is no less the chief excretion. * * * there is hardly a physiological process in which water is not of fundamental importance." The continued absence of liquid intake by man eventually results in the end of the life process.

According to Wolf,[4] "mean water intake by man per day is 2,200 g or 3.1 per cent of body weight per day." To be most useful to him, this daily quota of water must not carry contaminants antagonistic to his well-being; on the other hand, it should not be without those elements which contribute to his physical build-up.

Many living contaminants of water—the parasitic organisms which take up their abode on or within other living organisms to obtain food—are ready to attack man if they can get to him. Some of these use water as their habitat while others require it to complete their life cycle or as a vehicle to a point of entry to man. Some attack man directly; others need water-living intermediate hosts to produce their attack posture.

Water consumed by man may also contain a large number of non-living or chemical contaminants and a variety of them can be present in water both prior to and after its purification. Physiologic mechanisms of travelers and

newcomers to communities may be upset by a sudden change in water quality and if the mechanisms are impaired, the capacity of the body to adjust to these changes may break down. Some of water's chemical constituents like arsenic, cadmium, chromium, lead, selenium and nitrate are not known to be beneficial; contrariwise, at certain levels of concentration, they may produce serious, adverse physiological changes in man. Copper, iron and zinc may be metabolized with benefit as supplements to normal nutritional requirements. Excessive fluoride will cause dental fluorosis but, if present in an optimum amount, it will reduce dental caries. A deficiency of iodine is the primary cause of simple endemic goitre and hence a diet of water and food balanced to furnish the requisite amount of iodine is essential to good health. The sodium content of drinking water becomes important if a restricted sodium regimen is demanded for the control of several diseases of the heart, kidney and liver. Other new organic contaminants are reaching man through his drinking water but the physiological affect of most of them still remains to be determined.

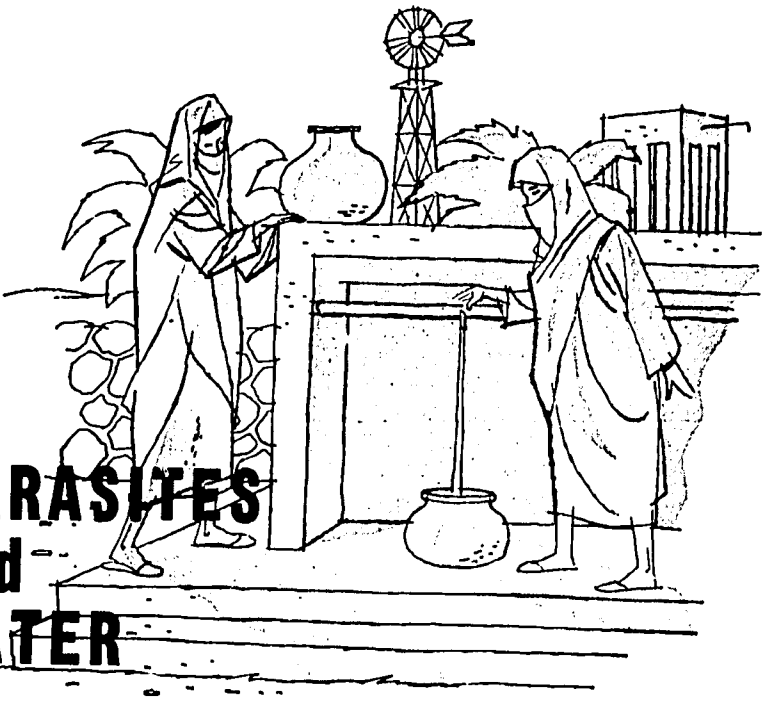
Man, as well as other living matter, is always exposed to background ionizing radiation coming from outer space and from traces of naturally radioactive isotopes, chiefly one of potassium and those of the radium series, in the soil and water. These are of low intensity and exert little significant effect on man during his lifetime. However, with the search for, and processing of, uranium ore and the employment of radioactive isotopes in a variety of useful ways, the effect of discarding wastes from these actions on water and the ground and secondarily on man, becomes a matter of great import. Artifically-produced ionizing radiation plus that naturally present must not be permitted to rise to a level inimical to man's health.

In a less complicated manner, water is important to man's health. The need to keep the body clean, to remove substances and organisms which can thoughtlessly or unknowingly be caught up in entrances to the body, is of great importance. The small child who rubs his itchy eye with a dirty finger has no realization that he may be pushing the trachoma virus into his eye. Given opportunity and encouragement to wash his hands, this might never happen. Cleanliness is inimical to external parasites such as lice and mites, and to the fungi responsible for skin diseases. The use and reuse of common utensils in homes and public institutions and places, without adequate cleansing and disinfection, is another cause of disease transmission even among people living in relatively highly advanced countries. Without adequate, safe water, conveniently available, many communicable diseases will just continue to run their disabling course. Dispelling filth, on the person and in the surroundings, requires clean water sufficient to maintain hygienic conditions.

REFERENCES

3. Henderson, L. J. The Fitness of the Environment. McMillan Company, New York (1913).

4. Wolf, A. V. Thirst. Charles C. Thomas—Publisher, Springfield, Illinois. (1958).



PARASITES and WATER

Parasites (in the broad sense), water and man are related in very specific ways. Many of these relationships result in recognizable disease * patterns each of which is characterized by distinct clinical and pathological features. However, rather than focusing attention on these features, it is intended herein to direct consideration to the links between the infecting agent, water and man. In some cases, the role of water is simply that of a vehicle of transmission; in others it plays a more physiologic or interstitial part. We have fairly precise information about this relationship in certain diseases (Group I); with respect to others, our knowledge about it is doubtful (Group II); and in the case of still others (Group III), it is merely suspected.[5]

The diseases selected to bring out this relationship in the first group are arranged according to the type of parasite only to facilitate an orderly discussion.

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* Dependence for nomenclature, infectious agents and synonyms has been placed upon Control of Communicable Diseases in Man published by the American Public Health Association.

Group I

1. Bacteria
   a. Cholera
   b. Dysentery, Bacillary (Shigellosis)
   c. Leptospirosis (Weil's Disease, Canicola Fever, Swineherd Disease, Mud Fever, Hemorrhagic Jaundice, Fort Bragg Fever, Rice Field Fever, Cane Field Fever)
   d. Paratyphoid Fever
   e. Tularemia
   f. Typhoid Fever (Enteric Fever, Typhus Abdominalis)
2. Helminths
   a. Dracontiasis (Guinea Worm Disease)
   b. Echinococcosis (Hydatidosis, Echinococciasis)
   c. Schistosomiasis (Bilharziasis)
3. Protozoa
   a. Amebiasis (Amebic Dystentery)
4. Viruses
   a. Hepatitis, Infectious (Epidemic Hepatitis, Epidemic Jaundice, Catarrhal Jaundice)

Group II

1. Diarrheal Diseases

These are a group of illnesses, having in common the symptom diarrhea. They constitute a major health problem in many countries of the world and a minor one in many others. Within this group are some physical disturbances having precise etiological definition, as indicated in the discussions of a number of them in Group I, and others without it. Due to the heterogeneous character of the group and the numerous viewpoints expressed regarding its content, a separate discussion seems to be justified.

Group III

1. Pleurodynia (Epidemic Pleurodynia, Bornholm Disease, Epidemic Myalgia, Devil's Grippe)
2. Poliomyelitis (Infantile Paralysis)

The two diseases in this group, in which viruses are the agents, have certain characteristics which cause one to be suspicious of the part water may play in their transmission. Neither, however, as of the present, has sufficient epidemiological evidence to support a water-borne theory of communication.

MODE OF INFECTION. Water is of primary importance in the transmission of cholera, leptospirosis, typhoid fever, dracontiasis, schistosomiasis and infectious hepatitis. The agents of infection in the water-borne cases of cholera, typhoid fever, dracontiasis and infectious hepatitis are ingested; those of leptospirosis and schistosomiasis reach the victim most often through the skin or by way of the mucous membranes.

Although there have been instances in which water has served as the conveyor to man of the agents of bacillary dysentery, paratyphoid fever, tularemia, echinococcosis and amebiasis, it is more likely that he receives these agents through other channels. The usual point of entrance to man of the causative organisms of bacillary dysentery, paratyphoid fever, echino-

coccosis and amebiasis when they are water-borne is the mouth; the tularemia bacillus more often penetrates through the skin or mucous membranes.

CHANNELS OF TRANSMISSION OF INFECTION. The route between the reservoir of some diseases and the susceptible person is quite direct; in others it is more devious. This is exemplified for most of the diseases discussed in this chapter as follows:

Amebiasis:	man	food, flies, contact, water — man
Bacillary dysentery:	man	soiled hands, water, food, milk, flies — man
Cholera:	man	water—man
Dracontiasis:	man	water—crustacean—man
Echinococcosis:	animals	food, water, soiled objects — man
Infectious hepatitis:	man	water, contact, food, milk, shellfish — man
Leptospirosis:	animals	water, food, contact — man
Paratyphoid fever:	man	contact, food, milk, shellfish, water, soiled hands — man
Schistosomiasis:	animals	water—snails—water—man
Tularemia:	animals	ticks, flies, handling infected animals, food, water — man
Typhoid fever:	man	water, food, milk, shellfish, contact — man

ILLUSTRATIVE AFFECTS OF INFECTION. Each agent of infection in the above groups of diseases produces characteristic reactions in a host. The reactions may be both objective and subjective. Some agents produce more violent or acute reactions than others, and some invade one organ of the body in preference to others. The course of infection in man varies according to the disease, to the immunity pattern of the host, and, among a variety of other factors, to the channel or route of infection.

A few examples will bring out these aspects of the infection chain. Each of the six diseases in Group I caused by bacteria produces an acute, systemic infection. With respect to cholera, bacillary dysentery, paratyphoid fever and typhoid fever, the principal organ involved is the intestinal tract. The infective agents of leptospirosis and tularemia are disseminated by the blood; in the former, the liver is the organ most often affected while in tularemia lesions may appear in the liver, spleen, lymph nodes, lungs and kidneys.

In the same group, helminths cause three diseases. In dracontiasis, the mature larvae of the guinea worm develop into mature worms in the viscera and deep somatic tissues. When the gravid female migrates to the skin, allergic reactions occur and at the skin surface a blister is produced. Echinococcosis results from swallowing the eggs of the causative worm which further develop within the host to a cystic stage. These cysts produce both a mechanical and a toxic effect on the liver and the lungs where most of them lodge. In schistosomiasis, after the cerceriae have penetrated the skin and matured, the females begin to produce eggs. While some acute damage results from migrating larvae, the main pathology is caused by egg-laying. Small blood vessels may be blocked, inflammation may develop around eggs and pseudo-abscesses formed. Other eggs may reach the intestinal tract or the bladder, or be carried by the blood to the liver or spleen.

The agent of infection of amebiasis is a parasite of man's intestinal tract. Ingested infective cysts pass through a number of stages in the host producing trophozoites which get into the submucosa of the colon and cause ulcers.

Infectious hepatitis is an acute viral infection. Following ingestion of the virus, it reaches the blood stream. The major pathologic lesions occur in the liver.

CHOLERA. Cholera is a serious, acute, bacterial infection of the intestines, the infectious agent for which is *Vibrio cholerae* (Cholera vibrio). Severity differs greatly, ranging from explosive epidemics to mild cases. Fatality in epidemics may be as low as 5 per cent or as high as 75 per cent.

The occurrence of cholera in the world has been well studied. This disease has probably been present in India since the earliest days of recorded history but there is little evidence that it spread from there until 1817. Since that year, epidemics have taken place in many parts of the world. Carried by caravans, cholera swept through China, Mongolia, Siberia and into Russia, and along the Ganges valley to Afghanistan, Russia, Turkestan and Persia. Following water routes, it reached the east coast of Africa and in the same manner went from Europe to the United States and South America and finally across land to the west coast of both Americas.

Like the tides, the disease ebbed and flowed during the ninteenth century in spite of the acquisition of the new knowledge by Snow, Pasteur and Koch.

There has been no cholera in Ceylon since 1919, in Iraq since 1931, or in Iran since 1939.[6] Since 1923, epidemic cholera has occurred outside of Asia only in Egypt in 1947 when 20,462 fatalities were reported.

Cholera is endemic in India and East Pakistan, and there may also be foci of endemicity in Burma, Thailand, Cambodia and China. In India, yearly deaths from 1900 to 1951 have been in either five or six figure numbers. In Thailand in 1945 and 1946, there were more than 8,000 cases. Cases and deaths from this disease as reported by the World Health Organization for 1956 are shown in table 1 and more recent reports on cases of the disease are included in table 2.

TABLE 1 *
Cases of, and Deaths from, Cholera, 1956 [7]

Country	Population	Cases	Deaths	Death Rate per 100,000
Burma		13	6	.....
Cambodia		1	1	.....
India	376,741,669		24,372	6.8
Pakistan				
East Pakistan	42,062,610		18,471	4.4

TABLE 2
Number of Reported Cases of Cholera [8]

Country	Year 1958	Year 1959
Burma	9	3
India	66,536	13,858
Pakistan		
East Pakistan	16,423	20,095
Thailand	11,597	7,777

Before the discovery of bacteria, the infectious agent of cholera was a matter of conjecture. By means of epidemiological research, Snow [9] collected and presented evidence proving that contaminated water played a role in its spread. The violent London outbreak of 1854 which he studied so thoroughly occurred in the vicinity of the Broad Street pump near Cambridge and Broad Streets. There were about 500 fatal attacks of cholera in ten days and all case histories showed that water from the Broad Street pump had been used for drinking. This source of water, as well as other limited water sources, had become contaminated with the infective agent.

Additional evidence accumulated by Snow pointed up the difference in cholera incidence in the houses supplied by the Southwark and Vauxhall Company, obtaining their water from a polluted section of the Thames River, and by the Lambeth Company which had recently removed its intake

* The vital statistics quoted in this monograph should not be accepted as absolute. There are still difficulties to overcome in many countries before population, morbidity and mortality data will be free of imperfections.

to an area in the Thames "quite free from the sewage of London". Snow commented that "the mortality in the houses supplied by the Southwark and Vauxhall Company was * * * between eight and nine times as great as in houses supplied by the Lambeth Company * * *."

Snow's investigation and his subsequent explanation of it convinced almost all authorities that drinking contaminated water containing an infective agent was, at least, one way in which cholera could be contracted. After Koch discovered cholera vibrio in 1883, much new knowledge concerning bacteria became available and Snow's exposition of the place of polluted water in the causation of cholera was confirmed.

Convincing proof of the presence of cholera vibrio in water consumed by humans in India who later died of cholera was given to the 1884 cholera conference by Koch. As reported by Pollitzer,[10] Koch said, "I succeeded * * * in finding the comma bacilli with all their characteristic properties in a tank which supplies water for drinking and household purposes for all persons living round it and in the immediate vicinity of which a number of fatal cholera cases had occurred. As was established later, the linen of the first cholera victim succumbing nearby had been washed in the tank * * *. On its shore there were 30-40 huts inhabited by about 200-300 persons and 17 of these had died of cholera."

The 1892 cholera outbreak in Hamburg, Germany, is a classic example of the spread of cholera by a waterworks system. This city took raw water from the Elbe River and pumped it into its pipe system without purification. How the initial infection of the river water took place could not be definitely established. Sporadic cases of cholera occurred for several weeks and then the explosive outbreak started. In a period of a little over two months, there were nearly 17,000 cases of cholera with 8,065 deaths. In addition to Hamburg, Altona, a separate but adjacent city, and Wandsbeck, a nearby suburban town, were involved. Altona, on the Elbe River downstream from Hamburg, used this river as a source of water but filtered it by the slow-sand process before it was used for drinking purposes. The Wandsbeck supply was a small lake. Altona and Wandsbeck suffered much less from this outbreak than Hamburg. Distribution of cases in these communities could be closely related to the water supply available to the residents.

"The presence of cholera in man is * * * an indispensable prerequisite for the contamination of water supplies, which then secondarily become the vehicle of the infection * * *." [10] Water taken for human consumption from irrigation channels, tanks, ponds, springs,[11] wells, and rivers, has been found to contain the contaminating agent causing cholera. The infectious agent reaches these waters in more or less devious fashion but man is the original source of them; he is also often the instrument through which they reach the drinking water in a direct manner as opposed to the indirect one.

Some observers hold that cholera vibrios persist for prolonged periods in contaminated water. Read and Pandit,[12] after examining many water samples from rivers, tanks, wells, lakes, marshes and streams in Bengal where cholera was present, found that the organism persisted for more than five days but not longer than 16 days. It was their conclusion, there-

fore, that in the maintenance of epidemics, it is likely that the persistence of the organism in water is not so important as are repeated contaminations of the water supplies. By experiment, the cholera vibrio has been found to multiply in sterilized river or well water, and to preserve its vitality in such water for several weeks.[13]

Foods grown in areas fertilized with night-soil (excreta), or washed in or prepared with, water containing cholera vibrios, can become carriers of the infection. For example, lettuce and celery are good potential carriers because vibrios can find a suitable habitat in the moist crevices of their leaves.[6] Fish and shellfish taken from polluted water for human consumption should be avoided; they have also been incriminated in the spread of cholera. Flies have been implicated as a mode of transmission of this infection.[14]

The part played by carriers in the spread of cholera has been studied by many over a long time. In 1952, Pollitzer [15] concluded that there seems no reason "to revise the opinion, held by most experts with experience in areas where cholera is endemic or frequent, that only 'acute' carriers, that is, individuals late in the incubation stage, those actually ill, and possibly also those in early convalescence, are instrumental in spreading infection."

Man's susceptibility to cholera infection is variable. Clinical manifestations of the disease do not always follow the ingestion of cholera vibrio. There is agreement, however, that if the disease does not appear following infection, it is due mainly to a nonspecific resistance rather than because of a specific natural immunity. Recovery from a clinical attack appears to provide some protection against the disease for several years. As stated by Maxcy,[13] "an attack of cholera does not necessarily confer protection against a subsequent attack. Nevertheless, second attacks within a period of a few years are uncommon."

The prevention of cholera is a problem of protecting man from himself. The human reservoir is able to supply an adequate number of causative organisms for the various vehicles of distribution to bring to man's mouth, the portal of entry. Cut these lines of communication—water, milk and other foods, flies—and the hope of the Second World Health Assembly,[16] to eradicate cholera, can be satisfied.

There are a number of preventive measures which can be instituted to end the spread of cholera. Among them, the provision of safe water in quantities adequate for drinking and culinary purposes, and the maintenance of a decent standard of cleanliness rank high. If a public water supply (one distributed through a system of pipes) is in use, the water provided must be treated to the degree that it is always free of cholera vibrios and the integrity of the delivery system of pipes must be such that the water reaches the consumer in an unimpaired state. Small drinking water sources not intended for use to supply piped systems must be of safe quality and so protected against contamination that the water cannot be degraded either through natural causes or by man's negligence.

With respect to water not intended for drinking; that is, water used for bathing, swimming, or washing of materials or objects, unless there is assurance of its freedom from contamination, the only safe procedure is to

stay out of it and not use it for any purpose which would offer even the remotest possibility that any of it will reach the mouth.

BACILLARY DYSENTERY. Bacillary dysentery is an acute bacterial infection of the intestine; it is one of the most important causes of death among the very young and the enfeebled old, and among people living in primitive conditions. The infectious agents which reach the gastrointestinal tract via the oral portal are various species of the genus *Shigella* among which the more important ones are *Shigella dysenteriae, Shigella sonnei, Shigella flexneri* and *Shigella boydii.*

This disease is world-wide in occurrence and has afflicted man since the beginning of history. Dubos [17] says that Herodotus blamed the defeat of the Persian army in 380 B.C. partly on dysentery. Descriptions by Hippocrates in the same period suggest that the disease was well known in Greece.

Bacillary dysentery appears in epidemics in the temperate as well as the tropical areas of the world although it is more frequent and violent in the hot countries.[6] Major outbreaks have taken place in India, Viet Nam, China, Japan, Ceylon, Malaya, the Philippines, Java, northern Brazil, Haiti, Panama, Puerto Rico, Egypt, Syria, Arabia, Jordan, Israel, French-speaking Equatorial Africa and the Republic of the Congo (Leopoldville). Distribution curves show a relationship between temperature and disease but it is believed that the level of sanitation in a particular area is a more important factor in incidence than temperature by itself. This disease is a "filth" disease and it has been demonstrated that when good sanitation measures are employed by authorities and their constituents, epidemics can be curtailed although sporadic outbreaks of limited extent will continue.

TABLE 3

Death Rates Per 100,000 Population [18]

Country	Latest Available Year	Causes: Typhoid Fever	Causes: Dysentery, All Forms
Bolivia	1954	2.9	16.7
Canada	1957	0.1	0.2
Ceylon	1956	3.5	6.4
Costa Rica	1957	1.5	5.8
Hungary	1957	0.2	1.3
New Zealand	1956	—	0.0
Portuguese India	1957	5.1	12.2
Thailand [7]	1955	7.0	22.0
United Arab Republic			
Egypt	1955	6.3	1.4
Union of S. Africa			
White population	1956	0.2	0.7
Colored "	1956	2.3	4.5
Asiatic "	1956	0.7	7.4
United States	1956	0.0	0.3

Explanation of symbols: —(dash) = magnitude negligible
0.0 = magnitude less than half of unit employed

TABLE 4

Death Rates Per 100,000 Population From Specific Causes in the Americas, 1956 [10]

Country	Causes		
	Dysentery, All Forms (Includes Amebiasis and Bacillary Dysentery)	Paratyphoid Fever (Includes Other Salmonella Infections)	Typhoid Fever
Argentina [a]	0.1	1.0	
Bolivia [a]	27.1	0.5	6.7
Brazil [b]	7.8	0.0	1.6
Canada [c]	0.2	0.0	0.1
Chile	1.3	0.3	2.3
Colombia	5.4	0.4	4.3
Costa Rica	5.2	0.6	1.6
Dominican Republic [d h]	5.9	...	7.2
Ecuador [d e]	7.8	0.9	25.4
El Salvador [d]	6.1	0.1	2.1
Guatemala [d]	85.6	0.8	10.2
Honduras [f]	11.0	0.1	4.0
Mexico [d]	16.7	2.6	11.3
Nicaragua	3.0	31.0	3.6
Panama	1.9	—	0.3
Paraguay [g]	4.4	0.4	0.8
Peru [a]	17.9	...	9.0
United States [d]	0.3	0.0	0.0
Uruguay [d]	0.0	—	0.9
Venezuela	5.1	0.3	1.1

Explanation of symbols:
... = data not available
0.0 = magnitude less than half of unit employed
— (dash) = magnitude negligible
[a]—Year 1953
[b]—Federal District and seven State capitals
[c]—Excluding Yukon and Northwest Territories
[d]—Year 1955
[e]—Data for Boletin Estadistico, Vol. 2 (1957)
[f]—Data from 1954-1955
[g]—Incomplete registration
[h]—An errata sheet withdraws these figures as inaccurate

Current vital data on bacillary dysentery per se are not generally available; it is most often included in statistics covering all forms of dysentery. Death rates for certain countries and specific years for the dysenteries combined are shown in tables 3 and 4.

Jails and asylums [20] have long been the sites of outbreaks of bacillary dysentery due undoubtedly to over-crowding, poor sanitation and low standards of personal hygiene, and hospitals [21] have also had their share of incidence. Military operations have always been accompanied by dysentery. In the American Civil War, the annual morbidity rate attributed to dysentery in the northern armies was 876 per 1,000 and the death rate 10.3 per 1,000.

Compare this death rate with 0.003 per 1,000, the rate in the United States in 1955 for bacillary dysentery and amebic dysentery combined (see table 4).

In civil populations, the relative importance of bacillary dysentery as a cause of illness and death can only be surmised because an unknown proportion of such diagnoses as diarrhea, dysentery unclassified, and diarrhea and enteritis are bacillary dysentery. Also, inapparent or unreported infections undoubtedly greatly out-number the clinical cases.

Although the dual etiology of amebic and bacillary dysentery was established in 1896, the two forms are still not distinguished one from the other in large parts of the world. This is reflected in the death rates shown in tables 3 and 4 which indicate that many deaths from the two diseases still take place in many world areas. Differential diagnosis depends upon the demonstration of the etiological agent and until facilities with which to make such determination are more widely and readily available, the mixing of these two dysenteries when reporting illnesses to the authorities will continue. Incidence of the two diseases in several American countries is given in table 7. In some countries, reported amebiasis is more prevalent than reported bacillary dysentery; in others, the statistical picture is reversed.

Shiga isolated the organism named for him and showed its etiological relationship to dysentery. During the next 30 years, Flexner, Sonne and Boyd also contributed to the process of identification of infectious agents as evidenced by the names attached to the other species of *Shigellae.*

Since these findings, many investigators have shown that the principal agent found in severe, acute diarrheal diseases is a member of the *Shigella* group. For example, McGinnes [22] studied diarrhea and dysentery in Henrico County, Virginia (U.S.) during a three-year period, 1930-1933. Among 176 families, 302 cases were found to have occurred, and complete clinical records and repeated stool examinations were obtained upon 295 of these. Upon clinical study of this series of 295 cases, 141 (48 per cent) were classed as dysentery and 154 (52 per cent) as diarrhea. Bacteriological examinations of the stools of those diagnosed as having dysentery showed that 69.5 per cent were positive for *Sh. flexneri* or *Sh. sonnei;* similar action with regard to those diagnosed as having diarrhea produced positive results for the same two *Shigellae* in 40.9 per cent of the cases.

Man is the reservoir of the infection and feces of infected persons are the source of the organisms which infect others. Contaminated foods and milk [23] have been incriminated as transmitters of this disease and flies [24] and ice [25] have been involved in outbreaks. The capability of the housefly, *Musca domestica,* to transmit dysentery bacilli in its feces and particularly its vomit-drop has been confirmed by a number of experimenters.[26] [27] Contaminated water has also been proven to be a source of infection but genuine water-borne outbreaks have been comparatively rare and usually occur through the medium of faulty plumbing or pipe connections, or because of failure to adequately protect the small privately-owned drinking water source against invasion by infected feces.

Cox [28] has recorded that in 1937 in New York State, there were eight outbreaks of bacillary dysentery of which only two were proven to be water-

borne and that in 1938, in the same area, there were 23 outbreaks and only four of them were water-borne.

Eliassen and Cummings[29] made an analysis of water-borne outbreaks which occurred in the United States from 1938 to 1945. These outbreaks were divided as follows:

Disease	Outbreaks	Cases
Dysentery	35	8,622
Gastroenteritis	198	101,339
Typhoid fever	99	1,359

Among a number of general statements evolving from this analysis were that (1) private water supplies were responsible for 70 per cent of the total number of outbreaks, and (..) the contamination of the water distribution systems via cross-connections and back-siphonage was responsible for 42 per cent of the total number of cases.

In a study of water-borne disease outbreaks for the decade 1920-1929 in the United States, including both typhoid fever and dysentery, Wolman and Gorman[30] ranked the surface pollution of shallow wells as the most important cause of pollution of drinking water.

At the Chicago and Alton Railroad shops in Bloomington, Illinois (U.S.) one of three cross-connections between the drinking water supply coming from the city and the railroad's industrial water supply of quite inferior quality proved to be a weak point in this dual system. The city water was protected at this cross-connection by one gate valve but when the pressure became higher in the industrial system than it was in the one carrying city water, the valve leaked and contamination was purveyed to water drinkers causing 300-400 cases of dysentery and 130 cases of typhoid fever.[31]

Another large epidemic involving 3,000 cases of bacillary dysentery was attributed by Kinnaman and Beelman[32] to the entrance of sewage into the water distribution system of Newton, Kansas (U.S.) through frost-proof hydrants and water-closet valves which became submerged in the sewage. Early in 1960, there occurred in and near Ravena, New York (U.S.) an outbreak of dysentery caused by *Sh. sonnei* during which there were about 1,400 cases in a population of 3,200. The infective agent was confirmed separately by two laboratories. Although it was concluded that the public water supply must have caused this outbreak, how it did so was never proven.[33]

The maintenance of a high level of *Shigella* infection in a given population group depends upon the transfer of human feces containing *Shigellae* from one person to another. If the quantity of *Shigellae*-carrying feces in circulation is reduced, the number of cases of bacillary dysentery will follow the same trend. One means of curtailing transfer of infected feces directly from one person to another is to cultivate and maintain high standards of personal hygiene; where high standards have come into being, the bacillary dysentery incidence rate has been lowered. Another important measure directed to the same end is the control of drinking water quality from source to consumer to exclude contaminants carrying *Shigellae*.

In the direct person-to-person transfer, carriers play an important role.

During the course of the illness, the patient will excrete viable *Shigellae.* This situation will continue on the average for a month and in some cases the organism may persist in the intestinal tract for a year or two. In the McGinnes[22] investigation, 625 apparently well people who were in contact with persons clinically ill with dysentery and diarrhea were examined bacteriologically. Of these, 110 (17.6 per cent) were found to be carriers of *Sh. flexneri* or *Sh. sonnei.* In families where there were one or more bacteriologically positive cases of clinical dysentery or diarrhea, 25.3 per cent of the contacts were found to be carriers; in families where no case was proven bacteriologically positive, only 9.3 per cent of the well contacts were found to be carriers.

In addition to transmission in water, the lack of water plays an important part in the person-to-person communication of bacillary dysentery and this role is a quantitive one rather than qualitative. Of all the measures employed in the hygiene of the person, washing certainly ranks first. That cleanliness of the person is intimately related to the lack of transmission of bacillary dysentery (and other diarrheal disorders) is strongly implied by the results of careful work done by a number of investigators.

In 1950, in Fresno County, California, Watt[34] investigated diarrheal diseases and concluded that the availability of water for personal hygiene "influenced" the prevalence rates of *Shigella.* Somewhat later, a similar study by Stewart[35] was made in Georgia and the findings implied that *Shigella* infection rates were highest where water was least available for personal cleanliness. Some of the data derived from Hollister's study[36] of migratory workers living in cabin camps are shown in table 5. From these and other data, the conclusion was drawn that the control of *Shigella* infections may be measurably helped by making water easily and readily accessible for personal hygiene.

TABLE 5

Number of Cultures by Type of Subcamp

Type of Subcamp	Number of Cultures	Per cent of Cultures Positive	Per cent of Families[a] Positive[b]
Cabin with faucet and shower and/or toilet	985	1.6	2.5
Cabin with faucet only	688	3.0	6.2
Cabin with no plumbing inside ...	4,438	5.8	11.0

[a] — Family is defined as all persons living in one household.

[b] — A positive family is one in which at least one child was found to be positive for *Shigella* during any one month.

Man is, in general, susceptible to bacillary dysentery but the disease is more common and more severe in children than in adults. Relapses and second attacks of the disease occur but, in the tropics, it is not uncommon that persons living in areas where the disease is epidemic tend to become immune to well-defined clinical attacks.

Bacillary dysentery can be prevented by keeping man from putting feces containing the infectious agents into his mouth. On a community basis, measures which will help to keep man from doing this include:

1. Sanitary disposal of human feces. This will decrease the occurrence of *Shigellae* on man's fingers and in foods and milk which can carry them to his mouth.

2. The provision of safe water, free of the causative organisms, for domestic use in the home or the place of living.

3. Supplying safe water in a quantity adequate to permit the cleansing of hands and fingers that otherwise might contaminate food and drink.

4. Abatement of fly breeding so that they cannot serve as mechanical carriers of the infection.

On the individual basis, the most important actions are:

1. The practice of habits of cleanliness to preclude getting the infectious agents in one's mouth or transmitting it to a vehicle which subsequently passes it on to another's mouth.

2. Exclusion of flies from the home or place of living.

LEPTOSPIROSIS. Leptospirosis is an acute, systemic infection. Acute illness may last from one to three weeks and there may be relapses. The fatality rate is low and it increases with advancing age. In severe cases, the death rate may reach 20 per cent. An organism of the genus *Leptospira* (Order—Spirochaetales; Family—Treponemataceae) is the infectious agent, and the more important species causing the infection are *Leptospira icterohemorrhagiae, Leptospira canicola, Leptospira autumnalis* and *Leptospira pomona* but other species can also be involved.[37] The causative organism of spirochetal fever (at one time known also as Weil's disease which is now a synonym for leptospirosis) was discovered by Inada and his associates in 1915.

The reservoirs of infection of one or another of the species of *Leptospira* are distributed all over the world; similarly, the disease prevails in all parts of the world. This disorder is probably far more common than has hitherto been suspected; its association with jaundice has tended to delay the recognition of the true prevalence of leptospiral infections. Yager[38] believes that infections of this kind in North America, for example, are "of far greater significance than has been attributed to them in the past."

Mortality figures on a world-wide basis are meager. Cases of the disease, as notified to health authorities in 1957 and reported to the World Health Organization,[39] are as follows:

Africa; four countries	259
America; three countries	52
Asia; one country	34
Europe; sixteen countries	449
Oceania; seven countries	313
Total	1,107

For the decade, 1949-1958, the Pan American Health Organization[40]

has gathered a small amount of information on the number of cases in a few countries, as follows:

Argentina	16
Barbados	1
Cuba	4
French Guiana	3
Haiti	1
Hawaii	18
Jamaica	9
Puerto Rico	100
United States	378
Venezuela	29
Total	559

Leptospirosis is primarily a zoonosis and man is an aberrant host. The reservoirs of the infection include cattle, dogs and swine, and rats and other rodents. Dubos [17] states that 40-60 per cent of the wild rats in the United States are naturally infected with leptospira. Urine of infected animals is the main source of the infection; rodents and common domestic animals will excrete the organisms in their urine for an indefinite period of time.[88]

The infectious agent presumably enters man through small breaks in the skin or through mucous membranes, or possibly as a result of the ingestion of contaminated water. The persons most likely to be attacked by leptospirosis are those who swim in contaminated waters; workers whose duties require frequent contact with contaminated water; [41] persons who work with fish, poultry,[42] and meats; [42 43] and those who live or work in premises heavily infested with rats. There is some belief that infected tissues also play a part in communicating the disease to man but Van der Hoeden [44] states that the "consumption of meat of infected animals will hardly cause any danger since, when rigor mortis sets in, the pH* of the muscles has dropped to a degree sufficient to destroy the leptospira."

The most common epidemiologic pattern is that in which man comes into contact with water contaminated with leptospira-containing urine of infected animals. Williams [45] described an epidemic of 24 laboratory-confirmed cases (22 children and two adults) of leptospirosis due to infection with *L. canicola* in a community in Georgia and two additional cases among non-residents. The source of the infection was a small swimming hole in a little creek. *L. canicola* was found to be in swine and cattle on surrounding farm land and in dogs in the settlement, all of which had easy access to the creek.

An epidemic of an influenza-like disease which attacked 50 of 80 persons who had been swimming in a creek near Geneva, Alabama (U.S.) was recounted by Schaeffer.[46] There were numerous rats and hogs on outlying farms. Eighteen of 22 of the involved persons showed antibody titer increases against *L. pomona*. A control group which had not been swimming was negative. In an Okinawa [37] outbreak, Gauld reported that 16 patients were shown serologically to have infections caused by *L. hebdomadis B;* ten of the 16 persons were swimming in an "off-limits" pond, two had had an

* Hydrogen ion concentration.

accident which trapped them for a short time in a rice paddy where they swallowed some of the water, and evidence was strong that the other four had also been swimming in the "off-limits" pond.

In the Soviet Union, Mikhailovskii [47] found that water played the leading role in the spread of infection in a new endemic focus of leptospirosis. It was serologically determined that *L. grippotyphosa* was the infective agent and that field mice were the most probable source of the leptospira.

Chang [48] studied *L. icterohemorrhagiae* with respect to its survival in natural waters and its reaction to disinfectants. In Charles River, Massachusetts (U.S.) water the survival time ranged from three to four days at 31-32 degrees C. to eight to nine days at 5-6 degrees C. It was also found that high and particularly low hydrogen ion concentrations (5.0 and 8.5) were detrimental to leptospira survival. At a hydrogen ion concentration (pH) of 5.0, calcium hypochlorite killed all leptospiras in one minute when the residual was 0.5 part per million (ppm) and in three minutes when the residual was 0.3 ppm. Similar figures at pH 8.0 were one minute—6 ppm residual; three minutes—3 ppm residual. Kirschner and Maguire [49] reached conclusions similar to those of Chang and his associates with regard to the influence of environmental conditions on the survival of *L. pomona.*

The susceptibility of man to this disease is general. Although agglutinating antibodies have been found to persist in the blood of recovered patients for many years, second attacks occur.

"Preventive measures vary according to (1) the ecology of the carrier host, (2) the mode of transmission of infections, and (3) the population at risk, and they must be developed to meet the different epidemiologic patterns encountered." [17] The chance of picking up an infection when swimming in artificial pools is relatively remote and it becomes more so when effective measures to exclude animals—particularly rats—from such pools are provided. In regard to the "old swimming holes," there is not much that can be done to reduce risk to swimmers in them except to warn them of it. When workers in sewers, mines, rice fields and other naturally wet areas must, by reason of their tasks, come into frequent contact with water subject to infection, protective clothing should be worn by the workers. This is true, also, of industrial employees handling meat and poultry. Rat-proofing of industrial establishments to keep out rodents will reduce the exposure risk.

PARATYPHOID FEVER. Paratyphoid fever is a generalized bacterial infection of man which is clinically indistinguishable from typhoid fever but is milder. The mortality rate of 1-5 per cent [50] is much lower than that for typhoid fever.

The agents causing this infection [5] are:

1. *Salmonella paratyphi*—a natural pathogen of man, not known to be a natural pathogen of other animals; [51]
2. *Salmonella schottmuelleri*—a natural pathogen of man but also found rarely in cattle, sheep, swine, lower primates and chickens; [51] and
3. *Salmonella hirschfeldi*—a natural pathogen of man.[51]

The number of serotypes of *Salmonella* has now become so large that they have been separated into groups.* *S. paratyphi* is included in Group A; Group B contains 51 serotypes including *S. schottmuelleri;* and Group C1 contains 49 serotypes among which is *S. hirschfeldi.*

Salmonella organisms are found in man in all parts of the world and in the intestinal flora of many domestic and wild animals. Group members of this organism are not, however, uniformly distributed throughout the world. For example, Group A is common in the Balkans, China, Egypt and Mexico; Group B is common in northern Europe and the United States; and Group C is widely distributed.

The disease occurs throughout the world in epidemics and sporadically. It is most likely to strike with greater frequency and intensity where effective sanitation measures have not been instituted. Undoubtedly there are many unrecognized infections as well as many clinical cases which are never detected or reported to authorities.

Global incidence of cases notified to health authorities for 1957 [39] are shown below:

Africa	298
America	7,619 [a]
Asia	2,800
Europe	13,918
Oceania	47

[a] Of this group, the United States reported 6,693 in which other salmonellosis is included.

Man is the reservoir of the agents causing paratyphoid fever as well as other *Salmonella* infections. Saphra [52] studied 9,284 clinical and subclinical human *Salmonella* infections at the New York Salmonella Center (New York City) and found that 98.1 per cent of them were in Groups A, B, C1, C2 and D excluding *S. typhi,* and E. In more detail for the first four of these groups, there were in

Group A	0.5 per cent
Group B	42.2 per cent
Group C1	28.8 per cent
Group C2	13.4 per cent

The sources of the infection are the feces and urine excreted by those having an acute infection or others who have recovered but are temporarily carrying the bacteria in the body. According to Rosenau,[13] at least 2-4 per cent of the cases of paratyphoid fever may be expected to be chronic carriers. In Saphra's [52] study of 7,779 cultures from human *Salmonella* infections, he learned that 15.5 per cent of the 18 species of *Salmonella* identified came from healthy carriers.

As is the case with other filth diseases, paratyphoid fever is communicated by man s transfer of infected dejecta to his mouth either directly or by means of a vehicle such as food or water. Milk and milk products and shellfish are suspected as being the main food vehicles. The short distance

* See Kauffmann-White classification (1955) in reference (51).

between man's contaminated fingers and his mouth lends force to the importance of proper and safe disposal of infected fecal matter and of personal cleanliness as means of severing the lines of communication of this disease.

Unlike typhoid fever, very few outbreaks of paratyphoid fever can be attributed to drinking infected water. Similarly, the indirect part that polluted water plays in contaminating shellfish and milk utensils, for example, is not too well substantiated by published epidemiological researches.

Evidence relating paratyphoid fever occurrence to the infective agents set forth above is not available but there are citations of a considerable number of cases due to *S. paratyphi A, B and C.* Whether these are specifically *S. paratyphi, S. schottmuelleri,* or *S. hirschfeldi* is not clear.

In 1946, *S. paratyphi B* was isolated from the water supply of Neuötting, Germany [53] following an epidemic of 415 cases of what was clinically named typhoid fever. A second water-borne epidemic of 816 cases due to the same cause was reported in 1950. Bursting water mains allowed organic matter to enter them which absorbed the chlorine added to the water making it ineffective.

An infection of a well through drains defective due to an unknown cause produced a water-borne paratyphoid fever outbreak in Brixworth, England in 1951.[54] *Bact. paratyphosum B* (*S. paratyphosum B*) was isolated from the water. Four cases showed clinical symptoms and 21 others excreted the infectious agent at one time or another though not all were positive on the first examination. In a small town in Slovakia in 1948,[55] 37 cases of paratyphoid A occurred. Wastes from a hospital, inadequately purified, were discharged into a stream used by many town people.

In Worcestershire, England in 1941,[56] a milk supply was apparently contaminated secondarily from a polluted brook water supply. In this outbreak, there were 17 primary and secondary cases of paratyphoid B, and "paratyphoid B bacilli" (*S. paratyphi B*) were isolated from the feces of nine of the cases. Another outbreak of paratyphoid fever involving 54 cases happened in the Carse of Gowrie, Scotland in 1944.[57] The vehicle was milk infected by water drawn from a brook. Paratyphoid B (*S. paratyphi B*) organisms were involved in the infection.

Man is generally susceptible to infection by *Salmonella* including those held responsible for paratyphoid fever. Some immunity, species specific, usually follows recovery from the infection.

Measures appropriate to reducing the spread of this disease may be broadly stated as:

1. Elimination of the sources of infection and of the modes and vehicles of transmission, and
2. Increasing the resistance of the susceptible host.

As preventive actions for paratyphoid fever are similar to those for typhoid fever, more detailed discussion of them will be made in connection with the latter disease.

TULAREMIA. Tularemia is an acute infectious disease caused by a bacterium, *Pasteurella tularensis.* This infection is basically one of wild

mammals; man is only an incidental host. Tularemia has probably been endemic in the Americas and Asia for a long time but in 1912 McCoy and Chapin [58] found it in dead rodents in Tulare County, California, and named the causative organism *Bacterium tularense*. The mortality rate in 15,525 cases reported in the United States from 1915 to 1942 was 6.9 per cent.[17]

Tularemia is present today in the Americas, in many parts of Europe, and in the Soviet Union, Turkey and Japan. Ordinarily, cases appear sporadically but the disease may become epidemic. Schmidt [59] has reported an epidemic of water-borne infection caused by the contamination of wells and streams by water rats. In the Rostov, Soviet Union area, there were 8,500 cases in November 1941 and 14,000 in January 1942, presenting a serious epidemic situation.

Data on cases of, and deaths from, tularemia are sparse but the relative reported prevalence in the Americas is illustrated by the following figures compiled by the Pan American Health Organization [40] for the years 1949 through 1958:

Alaska	2
Argentina	3
Canada	64
United States	7,052
Venezuela	52

At least 48 species of vertebrate animals have been incriminated as hosts of tularemia. In the United States, naturally infected hosts include rabbits, squirrels, mice, rats, beavers, chipmunks, opossums, skunks, quail, prairie chickens and pheasants. It has been estimated that 1 per cent of the wild rabbits in the United States are infected.[60]

When man becomes infected, the portal of entry is by way of the skin, mucous membranes, or the respiratory or alimentary tract. Contact by man with infected tissues or body fluids of infected animals, birds or insects is the most frequent cause of his infection. Simons [61] estimated that 81 per cent of the tularemia cases in California were caused by contact with infected wild jack rabbits. Other means of acquiring the infection are from the bites of flies or ticks, by ingesting contaminated food or water, or by inhaling dusts carrying the causative agent. Certain species of ticks, and one species each of the deer fly and the mosquito, are important reservoirs of tularemia; however, "survival in 54 arthropods has been reported." [17] Under natural conditions, this disease is not transmitted from man to man.

A serious epidemic involving 28 cases in a Civilian Conservation Corps camp in 1935 in northern Utah was reported by Hillman.[62] Many Corps employees worked stripped to the waist in warm weather and had many fly bites as a result of doing so. Deer flies had become much more numerous a week or two before the first case occurred. Jack rabbits were found dead without signs of having struggled and older men who were not outside working without shirts were free from infection.

Eating foods infected with *P. tularensis* has been proven to have caused tularemia in man in many instances. Amoss [63] reported two cases—both fatal—which appeared to be caused by eating infected rabbit. Francis [64]

described three family outbreaks resulting in 13 cases and eight deaths ensuing from the consumption of insufficiently cooked rabbit meat.

The first reported outbreak of tularemia due exclusively to water was related by Karpoff.[65] In 1935, 43 cases occurred among native hay-mowers in the Soviet Union.[66] It was shown that they drank unboiled water from a brook which was thought to have been contaminated by water rats. Another outbreak [66] in the Soviet Union happened when drinking water was taken from a small river which was a favorable habitat for water rats. This outbreak ended when the source of infected water was cut off. One confirmed case of this disease has been attributed to handling fish which came from waters undoubtedly carrying the infectious agent.[67]

Parker [68] and his associates reported the results of exhaustive studies of natural waters in the United States. He was able to demonstrate that water from streams in the northwestern United States, as well as the mud under the water, were positive for *P. tularensis* by the use of guinea pigs. "Repeated attempts were made to isolate *P. tularensis* directly from naturally contaminated water * * * but no definitely positive results were obtained." Contamination in the water and mud may be present at any season and may persist for at least 16 months. Quoting a Soviet Union source, Parker indicates that *P. tularensis* is said to remain virulent in damp soil for 30 days; in ice for 32 days; and in hydrant water for 95 days.

Humans of all ages are susceptible to tularemia infection but fortunately permanent immunity follows recovery from an attack.

Preventive measures are, of necessity, only general in character. When one is working, hunting or camping in an endemic area, care should be taken to avoid bites by, or handling of, flies or ticks. Rubber gloves should be worn when a killed wild rabbit is to be dressed or when performing laboratory experiments involving this animal. The meat of a wild rabbit should be thoroughly cooked before it is eaten. Raw water, in an area where the disease exists among wild animals, should be disinfected before it is consumed.

Public water supplies drawing upon sources likely to be contaminated with the infective agent can be made safe against infection by this disease by disinfecting the water with a hypochlorite or with chlorine solutions in water. Foote [69] conducted some chlorination experiments in a flowing stream under natural conditions and found in one case that *P. tularensis* was rendered harmless when the chlorine dosage was such as to give a residual chlorine content of 0.1 ppm after 15 minutes of contact; in a second trial, the same effect was produced on this agent after 30 minutes of contact with a residual chlorine content of 0.2 ppm.

TYPHOID FEVER. Typhoid fever is a systemic infection with a fatality rate of about 10 per cent. Typhoid-like illnesses were described in ancient times but the name was first used by the French in the early part of the 19th century. Typhoid fever and typhus were clinically separated by Gerhard in 1836 and Budd, by epidemiological research during the period 1855-1870, concluded that fecal discharges of infected persons were the source of the contagion. Finally, in 1880, Eberth isolated the typhoid bacillus.[60]

The infectious agent of typhoid fever is *Salmonella typhi* or typhoid bacillus, a pathogen found only in man. A dose of relatively few of the organisms is sufficient to cause the disease.

This disease is widespread throughout the world and is still a common one in many countries in Africa, South and Central America, Asia and Eastern Europe. The extent to which it is currently taking its toll is illustrated by the number of cases notified to health authorities in 1957 and recorded by the World Health Organization: [89]

Africa	27,145
America	45,273
Asia	29,588
Europe	51,727
Oceania	294
Total	154,027

Additional data on the number of deaths and on death rates are shown in tables 3, 4 and 6.

TABLE 6
Deaths by Selected Causes in Ceylon [70]

Cause	Year 1954	1955	1956	1957	Rate per 100,000 in 1957 [a]
Cholera	1	...	...	...	...
Dysentery [b]	621	730	570	754	8.4
Gastroenteritis and colitis [c]	4,337	5,420	3,688	4,204	46.7
Infectious hepatitis	63	65	124	184	2.0
Paratyphoid fever	9	8	2	3	...
Typhoid fever	362	313	309	234	2.7
					59.8
Ancylostomiasis	674	702	466	445	
Due to other helminths	3,894	4,092	3,392	3,411	
					42.8

[a] — Based upon a population of 9,000,000
[b] — Amebic and bacillary
[c] — Excluding diarrhea of the newborn

The only known natural hosts of *S. typhi*, the causative agent, are human beings, and the number of patients and carriers able to transmit the disease is large. Feces, and to a lesser degree, urine of infected persons are sources of the infection. Hence, either infected fecal matter or urine must be transferred from one person's intestinal tract to the portal of entry—the mouth—of another person to effectuate the transmission of the disease.

Communication of typhoid fever may be by direct or indirect contact with an infected person—either one who is ill or one who has recovered but still harbors the infectious organism. A number of vehicles of transmission have been incriminated in outbreaks and water and milk, among them, have been responsible for great epidemics of this disease. Other

vehicles of significant importance are raw fruits and vegetables, milk products, shellfish and occasionally other foods. The fly may serve as a vector by mechanically carrying *S. typhi* from feces to foods which are subsequently eaten.

Clark[71] has described a small, milk-borne outbreak involving 23 cases of typhoid fever taking place in 1938 at Great Brak River, South Africa. Three boy workers on a dairy farm became ill from an unproven source of infection and they were nursed by their female employer, the owner of the dairy farm. This woman milked the cows after the boys became ill. Excreta from the patients was disposed of in the kraal where the cows were milked. No sanitary conveniences of any kind were available on the dairy premises. All the families which used the milk were affected and in most homes there were multiple infections.

Illness due to eating foods carrying one or another of the many *Salmonella* are frequently reported in the literature. Typhoid fever attributed to the ingestion of infected common foods is not so frequently reported. In May 1960, an epidemic of 31 cases occurred in southern Louisiana (U.S.).[72] Twenty-five of 79 persons who attended a wedding reception and six of 19 who did not but who ate some of the same food served at the reception made up the group of ill ones. *S. typhi* were isolated from all the patients and from one woman who prepared the chicken salad sandwiches to which the spread of the disease in this incident was attributed.

A large outbreak of typhoid fever caused by eating infected oysters has been described by Lumsden and his associates.[73] In 67 cities in the United States, during a 60-day period late in 1924, there were estimated to be "over 1,500 cases of, and 150 deaths from typhoid fever in excess of the normal expectancy for such period." Definite epidemics of typhoid fever were evident in three cities and a marked excessive prevalence in ten. Epidemiological histories were obtained on 486 cases. Of these, 452 gave definite evidence of having eaten raw oysters and 77.2 per cent of this number (349) of having eaten them within 30 days prior to the onset of the disease. The main factor in the spread of the infection causing these epidemics was shell oysters and the preponderance of evidence indicated that the shell oysters were infected through the medium of water.

In 1937, in a South African village with a risk population of 190, there were 27 cases of typhoid fever and three deaths.[71] The source of the infection of the first case among these inhabitants could not be proven but the others, all within the same group of 190 people, were attributed to fingers, flies and filth. Houses were over-crowded and dirty, and food freely exposed to flies of which there was an abundance. Pails were used for excreta disposal and they were located close to the homes and freely accessible to flies. Water and milk as vehicles could readily be ruled out.

There is no paucity of written evidence relating typhoid fever outbreaks to the ingestion of impure water. Eliassen's[29] analysis of water-borne outbreaks in the United States from 1938 to 1945 showed that 99 of a total of 332 outbreaks were due to typhoid fever and that these accounted for 1,359 of the total of 111,320 cases of water-borne illnesses. Drinking raw

water from streams or rivers, dug or drilled wells, or other sources has frequently resulted in disaster to the consumers. Piped systems of water have, due often to unfortunate and unforeseen circumstances, also made their contribution to the typhoid fever toll.

In 1916, Ferguson [74] described an outbreak of 201 cases—some of which were admittedly of secondary origin—initiated at Old Salem Chautauqua grounds in Illinois (U.S.). Here water was taken from three inadequately protected dug wells. The sewage-polluted Sangamon River, forming one boundary of the grounds, overflowed and submerged two of the wells. Thirteen deaths were caused by this incident. As indicated heretofore, Wolman and Gorman [80] ranked the surface pollution of shallow wells as the prime cause of the pollution of drinking water following their study of water-borne disease outbreaks.

At Croydon, England,[50] about 40,000 members of the population received their drinking water from a well 205 feet deep. It became necessary to do some work on this well in 1937 through one of its adits. Among the workmen was one who later proved to be a chronic carrier of typhoid bacilli. Strict precautions to avoid the contamination of the water by the workmen were taken but they proved insufficient because 341 cases (including 19 secondary ones) and 43 deaths were attributed to this water supply which had not previously been implicated in an epidemiological incident. If chlorination of the water had not been in abeyance when work on the well was started, this disastrous outbreak might never have taken place.

The geographical spread of a large group of infected persons, providing opportunity for secondary infections, is sharply illustrated by an incident in 1958 at a religious assembly encampment in Missouri (U.S.) attended by more than 550 persons.[75] During the course of this ten-day meeting, more than half of those attending had diarrhea. A drilled well, 180 feet deep, slightly downhill and 160 feet from a septic tank and a sewage drainage area, furnished the drinking water. Following a heavy rain, water from the well was foul and was popularly condemned by the campers. After the encampment, the participants returned to their homes extending from Pennsylvania to California. Eventually 19 proven and 14 suspected cases of typhoid fever were traced to drinking the water at the encampment grounds. The attack rate among participants was estimated to be 6.2 per cent.

Faulty drinking water system operation resulted in an outbreak of great magnitude in Hanover, Germany, in the fall of 1926.[76] At this time there were 20,000-30,000 cases of gastrointestinal disease in a three-week period and this was followed in September by a total of 2,500 cases of typhoid fever within a 60-day interval of time. Only part of the public water supply was blamed for this catastrophe.

Failure to disinfect water adequately prior to its consumption—particularly water not receiving any other purification—has resulted in much illness and many cases of typhoid fever. At Xenia, Ohio (U.S.),[77] one of two sources of the public water supply is a mixed one of surface and ground water. Disinfection of this water was done by carefully dissolving dry bleaching powder and adding the solution at a predetermined rate. The bleaching

powder proved to be of inferior quality with a strength of only one-fifth of that for which it was guaranteed. During a three-week period, 44 cases of typhoid fever resulted from drinking this inadequately disinfected water. At Tonawanda, New York (U.S.),[78] in 1920, raw Niagara River water was used for drinking. The typhoid fever rate of this city had always been high but the outbreak reported here produced 236 cases in three months. Disinfection of the water with chlorine was started following the epidemic.

As recently as 1959, Keene, New Hampshire (U.S.) was struck with an epidemic of 14 recognized cases of typhoid fever.[79] A confirmed carrier of *S. typhi* working near a brook which found its way into the raw water supply; a period of heavy, washing rainfall; a deficiency in the effectiveness of one of the slow sand filters and the failure to disinfect the finished water prior to its consumption were the circumstances which combined to produce this outbreak. All 14 patients as well as the workman-carrier were found to have been infected with *S. typhi* Phage Type E following the use of the phage typing technique in the epidemiologic study.

Water serves frequently as the vehicle of transmission of typhoid bacilli. When sanitation principles are only crudely followed and infected excreta are thrown on the ground, the first washing rain will carry those pathogens that survive into the nearest water course. Then if purification of the raw water is not accomplished prior to use, the scene is set for more cases of the disease. Beard [80] studied for four years the survival of *S. typhi* in soils and he concluded that 50 per cent of them will perish in the first 48 hours but that the remainder may live over a period of months depending upon the amount of rainfall, the moisture-retaining power of the soil and the temperature. Morgan [17] says that "typhoid bacilli have been found to survive through an entire winter in frozen soil and for as long as seven days in well water." Houston's [50] studies of *S. typhi* survival in raw river water showed that five to nine weeks were needed to destroy them but that 99.9 per cent of them were gone in the first week. Shrewsbury and Barton [50] found that the same organism would live in tap water in daylight and at room temperature for 211 days. Hence, in unsewered areas particularly, the proper and safe disposal of infected excreta is a matter of great importance in the abatement of this disease.

As long ago as 1916, Johnson [81] urged that "the water works * * * should always be given preference in the allotment of moneys for the city's maintenance." Gradually this appeal was heard and water purification became a prerequisite to urban living. In 78 cities in the United States,[82] the decline in the incidence of typhoid fever, due largely to the elimination of unsafe drinking water, is portrayed by the following figures:

Year	Population	Death Rate per 100,000 Population
1910	22,573,435	20.54
1920	28,244,878	3.85
1930	34,386,717	1.61
1940	35,895,638	0.48
1946	35,895,638[a]	0.15

[a] 1940 census figures

The same encouraging reduction in typhoid fever incidence has taken place in some other world areas, as for example, Germany. Bruns,[83] in discussing typhoid fever in Prussia in 1933, concludes that faultless drinking water supplies have become the most important factor in preventing cases of it. He supports his contention with these figures on cases of, and deaths from, typhoid fever in Prussia:

Year	Cases per Million Population	Deaths per Million Population
1913 [a]	246	25
1920 [a]	440	38
1930	92	9

[a] Until 1925, typhoid fever and parataphoid fever were not distinguished from one another.

Where modern sanitation methods are not yet widely applied, epidemics still occur but generally, in those countries which exploit available sanitation and water purification knowledge widely, outbreaks are now sporadic and the principal source of infection is the unrecognized carrier of the infective agent.

Less than 5 per cent of typhoid fever patients can be expected to become chronic carriers and continue to serve as dispensers of *S. typhi* until detected and brought under supervision and control. Morzycki [84] has reported from Poland that over 19,000 persons holding various occupations were examined and 0.2 per cent of males and 0.4 per cent of females were found to be harboring the typhoid bacillus. Of some thousands examined after an attack of this disease 1.3 per cent of the males and 2.0 per cent of the females were excreting the organism after two years. Of 3,356 domestic contacts of typhoid fever cases, 5.3 per cent proved to be carriers. Hence, the carrier who may continue to excrete the causative organism for months or years after clinical recovery is the greatest single factor in the spread of typhoid fever in communities where adequate health services have been established. In rural or underdeveloped areas, the application of good sanitation principles still remains the most positive approach to the abatement of this disease.

The susceptibility of man to typhoid fever is general although there is some evidence to indicate that adults acquire a degree of immunity through unrecognized infections. Fortunately, a high degree of resistance usually follows recovery from an attack.

Since water is a significant vehicle of transmission of *S. typhi,* it is of utmost importance that when it is used for drinking it be free of viable organisms of this variety. Protection of a source of supply against invasion by this organism, purification of the supply by artificial means or both measures are needed. In the case of the small, non-piped water supply, protection of it against infection is often the only safeguard provided. Under some circumstances, this is adequate but a secondary precaution can be taken by boiling the water before using it. When piped water is available to consumers, purification—particularly disinfection with a chemical—is most often employed. However, artificial purification should not discourage the continuance of alert efforts to exclude contaminants from the source of the

water to be purified. There are many accepted and proven systems of purifying water to make it safe to drink; the most suitable to the situation should be selected following a consideration of all factors.

In the treatment of large quantities of water intended for distribution through pipes, the use of a bactericidal agent such as chlorine serves two purposes. It not only kills bacteria prior to the water leaving the purification works but it can be used to produce a chlorine residual in the system of pipes, thereby affording some protection against contaminants which may reach the water during its distribution. The amount of residual chlorine needed to insure satisfactory bacterial quality of drinking water varies with the type of water, the temperature, conditions of treatment, and the type of chlorine residual maintained.[84]

With free available residual chlorine in water at pH 6.0 to 8.0, a safe residual for complete killing of bacteria at the end of 10 minutes of contact time is not less than 0.2 ppm. On the other hand, with combined available residual chlorine in water at pH 6.0 to 7.0, a safe residual would not be less than 1.0 ppm for 60 minutes contact time.[85]

Other measures of significant importance in curtailing the incidence of typhoid fever are the sanitary disposal of human excreta to reduce the opportunity for it to reach man's mouth via fingers, foods or flies; the pasteurization or boiling of milk to keep it safe to consume as fluid milk or as a milk product; the control of shellfish-growing waters to provide a clean and safe habitat for their cultivation; and the detection and supervision of carriers of the causative agent. The individual can contribute to his own safety by avoiding the eating of raw foods unless they are throughly cleansed in water free of typhoid bacilli, by keeping flies from the home and particularly the foods, by contributing to the sanitary disposal of excreta, and by maintaining a high level of personal cleanliness.

DRACONTIASIS. Dracontiasis is an infection caused by *Dracunculus medinensis,* a nematode or true roundworm, commonly called the guinea worm. This worm has been known since ancient times and the infection it causes was recognized by Greek and Roman physicians.[86]

Guinea worm disease occurs in India, Pakistan, Afghanistan, Turkestan, Saudi Arabia, Iran, North and Central Africa, and the Soviet Union. No autochthonous case in man has been reported in North America. Stoll[87] estimates that there are 48,000,000 people in the world affected by it.

Incidence varies greatly. Quoting several sources, Gradwohl[88] states that in 1945 incidence in India ranged from 6 per 100,000 in Bengal to 3,964 per 100,000 in Mysore and West Madras. In one district of Bombay, 10 per cent of the population had the infection each year and in many villages 25 per cent of the people may be affected by it. Infections have been known to run as high as 95 per cent in groups of people as large as 15,000. "Although dracontiasis is not a fatal disease or one which attracts much sympathy, its incidence nevertheless is commonly the precursor of much suffering and physical incapacity."[89] Incapacitation may continue for five to six months and even as long as eight months if there are other physical involvements resulting from or accompanying the infection.

The adult guinea worm develops to maturity in the human body and when the female becomes gravid, she migrates to the skin where an ulcer is formed. If the open ulcer comes into contact with water, the female discharges larvae into the water. These wriggling larvae attract an appropriate species of *Cyclops* (Order—Copepoda; Class—Crustacea) which swallow them. The *Cyclops,* in turn, is swallowed by man in drinking water. In man's body, the worms are released from the *Cyclops,* penetrate the wall of the intestine, and live in the connective tissue of the body. In 8-14 months the females are ready to release the young. Larvae will remain alive in clear water as long as six days, and in muddy water or moist earth, they may live two to three weeks. *Cyclops* is widely distributed throughout fresh and salt water and most of them appear in still water. At least 12 species of *Cyclops* have been identified as being intermediate hosts of *D. medinensis.*

The susceptibility of man to dracontiasis is universal. No immunity is acquired following an attack; in fact, a person may have repeated and multiple attacks.

As has been explained, the method of transmission of this infection is a relatively simple one. Water is essential and preventive measures can therefore be aimed at this medium of transmission. As the perpetuation of the life cycle of the guinea worm requires the release of embryos in water, excluding infected persons from water sources—particularly those used for drinking—will curtail the infection of *Cyclops,* preventing transmission even though *Cyclops* is imbibed. The worm appears in the human more frequently on the feet and ankles (32 per cent), and the legs (50 per cent) than on other parts of the body.[86] These locations are most suitable for embryo release if the infected persons are either wading or working in water.

Measures to destroy *Cyclops* have been tested with considerable success. Pradhan[90] applied lime (CaO) of 80 per cent strength to wells at a rate of one drachm * per gallon of water. This optimum dose, used four to six times during the period from February through May, kept wells free of *Cyclops.* Ramakrishnan[91] obtained an excellent kill of *Cyclops* in laboratory trials using 50 per cent wettable DDT (dichloro-diphenyl-trichloro-ethane) in a suspension of 0.5 per cent strength.

Other experimenters, including Moorthy,[92] have tested the usefulness of small plankton-feeding minnows in keeping down copepods. Several small fish are useful for this purpose but they have their limitations and are not as effective as is required for total destruction of the *Cyclops.*

Dracontiasis can be prevented by keeping man from swallowing infected *Cyclops.* Hence, the provision of potable water—water free of living, infected *Cyclops*—is of first importance among preventive actions. Abolition of step-wells and measures to prevent the contamination of drinking water by the immersion of affected parts of infected persons are essential. Boiling water will kill *Cylops* and they can be strained from it by filtering through muslin cloth. Application of sufficient DDT wettable powder to give a concentration of 5 ppm, every six weeks, will keep *Cyclops* under control,

* The drachm is a dram which in apothecaries (Troy) weight is 3.88 g or 3.69 ml.

but the use of such a persistent insecticide in water to be consumed is not desirable.

Lastly, the education of the public to drink only boiled or filtered water, and in the mode of spread of the infection is required.

ECHINOCOCCOSIS.* One class of the platyhelminthes (flatworms) is the cestodes or tapeworms and among the members of this class is *Echinococcus granulosus* which is the cause of the infection, echinococcosis.

Echinococcosis or hydatid disease was well known to ancient writers on medicine. The Talmud mentions this condition in sacrificial animals, and both Hippocrates and Galen referred specifically to the disease.[93] Many investigators have studied the causative worm and the disease over the years to accumulate the information now available about them.

This disease is fairly common in southern South America, New Zealand, eastern Australia and countries of the Middle East. It occurs relatively frequently in the Indian population of Canada and the Eskimos of Alaska. Based upon the distribution of the larval stage in man and animals, it can be considered to be a disease of sheep- and cattle-raising sections of the world. The number of cases reported by the Pan American Health Organization[40] for five South American countries for the period 1949 through 1958 totals 4,226, with Argentina reporting the largest number.

The dog is the common definitive host and the main reservoir of the infection although other carnivores like the wolf, jackal and cat are important in this role also. The dog—especially those used in herding domestic animals—acquires the infection usually by eating offal and carcasses of infected intermediate hosts such as sheep, cattle, hogs and goats; the infection has also been found in many mammals other than these. Illustrative of the magnitude of the source of infection of the definitive hosts are the data assembled by the Pan American Health Organization[40] for the three years 1956-1958, on the reported cases of hydatid disease in animals in two South American countries where sheep- and cattle-raising are important industries; Argentina—1,088,878 and Chile—296,206.

Man, an accidental host, acquires echinococcosis by ingesting the eggs of *E. granulosus* which have been deposited in the feces of the infected dog or other definitive host. The ova reach man's mouth by way of contaminated fingers, or food or drink served in fouled containers or consumed with contaminated utensils. Only the larval stage of the parasite is found in man.

Eggs of the parasite will remain alive for weeks in moist and shady places but they will expire rapidly if exposed to direct sunlight.[86] As the eggs are non-resistant to high temperature, this disease consequently occurs more frequently in temperate than tropical climates.

"Direct contact is practically the only method of infection in human cases"[94] although the possibility of man's becoming infected from soil or vegetables or food cannot be ignored. Heinz[94] has shown that flies have the ability to transmit eggs of *E. granulosus* to foods. He conducted experi-

* The September-October, 1961 issue of World Health, the magazine of the World Health Organization, includes contaminated water as a mode of human infection of this disease.

ments which indicated that they will carry only a few eggs on the outside of their bodies but that a much greater number are ingested and that they can be recovered from milk after the flies have had access to it. The fly-ingested ova did not lose their viability during their movement. Water plays a very minor part in the transmission of the disease. Uncleanliness and the lack of hygienic practices set the stage for the human consumption of this parasite's eggs and the initiation of the major portion of human infections.

Man is universally susceptible to echinococcosis and there is no evidence of any acquired immunity subsequent to an infection.

Preventive measures on a wide scale are mainly directed to the elimination of the infection in the common definitive host, the dog. Tight control of the slaughtering of herbivorous animals to keep dogs from eating scraps of uncooked meat; the reduction in the number of dogs, specifically stray ones, in endemic areas; and the burning or deep burial of dead animals will go far to reduce the incidence of this disease. The public, with emphasis on children, must be educated to shun close association with dogs in areas where the disease is widely prevalent. By legislative and educational actions, Iceland was able to reduce an incidence of one in every 40 persons to one in 2,000 over a period of 20 years.[95]

With respect to sporadic human cases, avoidance of contamination of the person, and of food and drink, with dog's feces by keeping oneself and his surroundings clean will cut the chance of transmission. Boiling water will kill the eggs and make it safe to drink.

SCHISTOSOMIASIS. The fluke, a parasitic worm of the class Trematoda (Phylum-Platyhelminthes), is the causative agent of schistosomiasis, one of the most chronic of diseases. Three species of this trematode have been identified as the chief producers of this disease: *Schistosoma mansoni, Schistosoma japonicum* and *Schistosoma haematobium.* The first two give rise to intestinal and the third to urinary manifestations. Relatively recent information indicates that *Schistosoma bovis,* a blood fluke of cattle, has been found in man.[96] In some irrigated areas where cattle are numerous and they come into close contact with humans and use and contaminate the same waters, as many as 40 per cent of the human population have become infected.

Schistosomiasis may cause years of severe debility. Direct mortality from it is low but predisposition of chronic cases to attack by other diseases causing death is of great moment. The economic effect of the disease in areas where it is endemic is incalculable.

Early Egyption records and its discovery in Egyptian mummies of the period 1250-1000 B.C. show that schistosomiasis is a very ancient disease. It was observed in the French army in Egypt about 1800 but the parasite was not related to the disability until 1851 when Bilharz proved the connection between the parasite and its eggs to dysentery among the natives.

As schistosomiasis is not a notifiable disease in many countries of the world, specific data showing its current distribution are not available. It is known that *S. mansoni* occurs in Africa, the Arabian peninsula, north-

eastern and eastern South America and the Caribbean area; *S. haematobium* in Africa, the Middle East, Portugal, Western Asia and to a small extent in India; and *S. japonicum* in China, Japan, the Philippines and the Celebes.[5] The larvae of certain other schistosomes of birds and rodents penetrate the skin and cause a dermatitis often called "swimmer's itch." These schistosomes, non-maturing in man, are found in many parts of the world.

In most of the areas harboring the causative agents, the disease is endemic; Stoll [87] has estimated that 114,000,000 people in the world are afflicted with schistosomiasis. In some regions in East Africa, 50 per cent of the population may be infected with *S. haematobium*. Sullivan [97] made an epidemiological study of an outbreak of 575 cases of schistosomiasis caused by *S. japonicum* among American engineer and infantry troops during a five-month period in 1945 on the island of Leyte in the Philippines. The attack rate of those whose tasks called for work in water regularly varied from 71 to 89 per cent in different platoons.

Man is the principal host of *S. mansoni* and *S. haematobium;* for the most part, he passes the eggs of the former in his feces and of the latter in urine. *S. japonicum* is parasitic in a number of mammals such as dogs, pigs, water buffaloes, horses, sheep, field mice and wild rats, and it has been present in man. From these hosts, the eggs are released in the feces.

When eggs from any of the three parasites reach fresh water, they release free-swimming larvae or miracidia which must penetrate an appropriate snail, the intermediate host, within 48 hours or perish. Snails of the following genera have been incriminated as intermediate hosts: [98]

1. *S. mansoni—Australorbis, Biomphalaria, Tropicorbis*
2. *S. haematobium—Bulinus, Physopsis, Planorbarius*
3. *S. japonicum—Onocomelania*

After the miracidium has developed further within the snail, larvae or cercariae emerge ready to infect man or another mammal by penetrating the skin and to initiate the life cycle again. As the parasite does not multiply in the human body, severity is proportional to exposure. Less frequently infection will occur as a result of the penetration of the mucous membrane of the mouth by cercariae. The life of larvae is short, probably not extending beyond 48 hours, but a single snail can release many thousands of cercariae.

The susceptibility of man to infection with this disease is general. Some resistance develops as a result of infection but it is incomplete and reinfection is possible.

The life cycles of the schistosomes are quite well understood and the places of man and other mammals, and snails in these cycles are known. This understanding points the way to the perfection of preventive actions suitable to alleviate and preclude infections. Knowledge now available is such that this disease can be prevented in any localized area. The main deterrent to doing this is the difficulty of bringing together, for effective joint action, the skills, materials and effort.

The exclusion from fresh water of feces and urine of infected humans and other mammals will keep the agent's eggs from reaching the medium

essential to their development. With respect to man, this can be accomplished by the application of good sanitation practices, a difficult feat in many parts of the world where the value of sanitary disposal of human wastes is little understood; as regards infected wild mammals, exclusion from natural waters presents a problem of great magnitude although ponds are being fenced in a project in Ghana to restrict water use by domestic animals as well as humans. Provision of home-type facilities for sanitary sewage disposal will have to be accompanied by education in their use and there will have to be modification of life-time habits and cultural patterns to keep humans from having contact with water-carried infected feces and urine. With regard to sewage collection by pipes and its treatment in purification works, Jones [99] found that ova of *S. japonicum* are destroyed or reduced in number by primary sedimentation during the usual detention periods, anaerobic sludge digestion and intermittent sand filtration but that they would pass trickling filters and hatch in activated sludge.

Avoidance of infected waters affords complete protection against cercariae. Agricultural and other workers who must enter infected water can obtain partial protection against penetration of the skin by cercariae by wearing protective clothing. When Sullivan [97] made his epidemiological study in the Philippines, it was his preconceived thought that men in signal construction units would show a substantial portion of the total cases because in performing their duties they were exposed a great deal to water in ditches and swamps. This did not prove to be the case however. He could conclude only that their clothing and boots had protected them against cercariae. Further, the attack rate among infantry troops was only 0.5 per cent even though these men operated in flooded terrain in a highly endemic area. Boots, leggings and clothing must have protected them although this was not proven. There was no information available on the number of troops wearing clothing impregnated with dimethyl phthalate. Unfortunately, however, neither a prohibition against wading, bathing or working in infected waters nor the donning of protective clothing when entering such waters are enforceable edicts or are procedures generally followed.

A domestic water supply free of snails, snail eggs, miracidia and cercariae to use for drinking, bathing and household washing tasks would, at least, free from the possibility of infection those persons whose necessary duties do not include working in infected waters. An efficiently operated water purification plant terminating with adequate disinfection will produce such a safe domestic water. The flocculation process usually included in a water treatment system is by itself inadequate to insure the removal or death of cercariae of schistosomes even when aluminum sulfate alone or with lime pretreatment is used in the maximum quantities generally employed.[100, 101] Standard filtration through sand likewise fails to remove all cercariae.[101] Various forms of chlorine [101] applied to water which had been clarified and filtered through filter paper and which had pH values ranging from 7.5 to 8.9 gave satisfactory results against cercariae as follows:

1. Gaseous chlorine dissolved in water will kill with certainty in 1¼ hours when the initial concentration of available chlorine is 0.4 ppm

or more. If the contact period of 30 minutes, 0.8 ppm is needed.

2. Sodium hypochlorite is effective after 1⅔ hours when the initial concentration of available chlorine is 0.4 ppm. More is needed—1.0 ppm—when the contact period is shortened to 30 minutes.
3. Chloramine kills in 1⅙ hours when the initial concentration is 0.36 ppm. When only 30 minutes contact is permitted, 0.45 ppm of available chlorine is required.

Jones [100] states that there is no indication that cercariae of the various species differ significantly in their resistance to chlorination.

In additional work, Jones [102] learned that the miracidia of *S. japonicum* were killed within 30 minutes by chlorination when the terminal residuals were 0.2-0.4 ppm; chloramine solutions were effective under the same conditions when the chloramine residuals were 0.3-0.4 ppm. The ova of *S. japonicum* were killed within 30 minutes only when the 30-minute chlorine residual varied from 3.9 to 11.0 ppm. No ova of *S. japonicum* survived in one test with a chloramine concentration of 1.9 ppm and a two-hour residual of 0.5 ppm.

Water management, and the changing and improvement of land use and agricultural practices, are other methods of approach to the control of the transmission of schistosomiasis. In building dams to impound water for multiple uses, consideration must be given during planning and construction stages to ways and means of reducing the creation of habitats favorable to snail production. Excess water often contributes to health problems, and drainage and filling have a place in the reduction of snail propagation. Irrigation systems, now in wide demand in many water-short countries, are of particular importance because each new one may increase the potential snail-breeding area unless snail control accompanies its construction. Water-users still employing primitive irrigation and agricultural methods are major contributors to the continued production of snails and the maintenance of foci of schistosomiasis.

At the Miwani Sugar Estates in Kenya,[96] where sugar cane is irrigated, *S. mansoni* and *S. haematobium* are endemic in the surrounding area. Well-designed distribution and drainage systems on properly graded land, conservation of water by using pipes, cycling of irrigation periods, mechanical maintenance of the irrigation system and the use of herbicides have resulted in good crop yields and little or no snail breeding. These preventive steps are possible, however, only when control is centralized and crops have a high commercial value. At the Kpong Experimental Farm [96] in Ghana, mechanical rice cultivation procedures have been devised which upset the snail and parasite cycles and thus reduced schistosomiasis transmission.

Barlow [103] investigated the value of irrigation canal clearance and has proposed it as a useful measure in Egypt because it is efficient, inexpensive and requires no new operational procedures involving the training of workers. Clearance aligns the canals, frees them of weeds on which snails feed and reduces the number of snails appreciably. Barlow points out,

however, that two intermediate snail hosts of *S. haematobium* and *S. mansoni* in Egypt are able to withstand long periods of drying without losing their reproductive powers.[104]

Experiments by Chernin and his associates[105] have shown that *Marisa cornuarietis,* a large operculate snail, can limit the growth of populations of *Australorbis glabratus* under certain laboratory conditions. *Marisa* is a ravenous herbivore and while feeding on vegetation, it consumes *A. glabratus* egg-masses; it is also capable of destroying newly-hatched *Australorbis.* In the laboratory, repeated exposures of *Marisa* to the miracidia of *S. mansoni* have failed to produce infection. The usefulness of *Marisa* in controlling *Australorbis* is being studied further in Puerto Rico.[106] Under suitable circumstances, other natural enemies and predators may be of help in reducing snail populations.[107]

In the opinion of the Second African Conference on Bilharziasis,[96] molluscicidng offers the most efficient single method for controlling the disease. Copper sulfate and sodium pentachlorophenate have been used to kill snails for a number of years and other compounds, although now used mostly experimentally, show promise of usefulness. In Egypt, copper sulfate used at the rate of 0.6-1.0 ppm in standing water and 10-20 ppm in flowing water proved effective. In some experiments with sodium pentachlorophenate, a concentration of 6.0 ppm for six hours was successful.[108] The selection of the most suitable poison to use must be based upon costs, the condition of the waters to be treated, the uses to be made of the water by the local population and the local agricultural practices. The level of knowledge pertaining to molluscicidng is now such as to warrant larger scale operations in the use of this potent weapon to destroy the snail and reduce the amount of transmission.

Mass treatment of infected persons in endemic areas can help to reduce transmission but it has not aided materially in lowering prevalence.

AMEBIASIS. Amebiasis, manifesting itself in man primarily in the colon, results from infection by a protozoan of the species *Entamoeba histolytica,* the only member of the *Entamoeba* genus which is pathogenic to man.[86] The disease is only infrequently the direct cause of death.

In 1875, Lösch reported finding "*E. histolytica* in the feces of a Russian with severe dysentery" and "demonstrated its pathogenicity by infecting a dog which developed dysentery and intestinal ulcerations."[86] The ameba was related to the disease in 1887, and by 1913 the pathogenicity of *E. histolytica* and the current concept of the host-parasite relationship were established beyond doubt.

Amebic infection is world-wide, often to the extent of 50 per cent of the population in localized areas with primitive sanitation. Clinical amebiasis is prevalent in hot countries, and not so frequent in temperate zones although infection rates may be high. The prevalence in any population is likely to parallel the level of sanitation existing in the area.[109] Symptom-free infections may last for many years.

Klatskin[110] has reported that surveys among American troops serving in India showed infection rates between 20 and 40 per cent. Hegner and his

associates[111] examined 866 persons from two schools in Mexico City and six other Mexican communities and found the incidence to be 25 per cent. According to Sapero,[112] amebiasis incidence among United States naval recruits from southern states was 14.7 per cent and among those from northern states, 7.8 per cent. An examination was made of 1,060 freshmen of a United States college by Wenrich [113] and he found an incidence of 4.1 per cent of this disease. Based upon surveys of the inmates of 17 mental institutions, Burrows [114] reports an average of 25.7 per cent of them as harboring *E. histolytica*. Burrows also studied the results of 166 surveys of population groups totaling about 250,000 persons. His conclusions were that previous estimates of amebiasis prevalence have been too high and that *E. histolytica* is harbored by only 1.0 per cent or less of the population of Alaska and Canada, and by less than 5.0 per cent in the United States.

Recent information on the cases of clinical amebiasis reported to the national health service of each country in the Americas has been gathered by the Pan American Health Organization [40] and is as follows:

1949	81,315	1954	130,705
1950	85,095	1955	112,445
1951	84,078	1956	111,713
1952	100,749	1957	120,278
1953	118,752	1958	141,518
		Total	1,086,648

Case rates for this disease in certain countries in America are set forth in table 7.

TABLE 7
Reported Cases of Amebiasis and Bacillary Dysentery per 100,000 Population in the Americas, 1958 [40]

Country	Cause: Amebiasis	Cause: Bacillary Dysentery, and Other Unspecified
Argentina	17.8	21.9
Brazil	...	25.3
Canada	0.0	9.1
Chile	...	6.9
Colombia	623.4	92.7
Costa Rica	82.7	26.7
Dominican Republic	83.1	91.3
Guatemala	...	113.8
Honduras	558.5	364.6
Mexico	75.6	14.6
Panama	32.2	130.8
Paraguay	...	127.4
Peru	...	290.9
United States	2.5	6.8
Venezuela	502.1	2,205.0

Explanation of symbols: 0.0 = rate less than 0.05; . . . = data not available

The reservoir of the infectious agent is an infected human, usually a chronic case or one without obvious symptoms. Animals are of little significance in the transmission of this disease to man. The source of the infection is cysts in the feces of the infected person.

The mode of transmission of amebiasis is not a complicated one. Man, infected with the ameba, passes the mature cysts in his feces. By way of food, contact, flies, carriers and water, the cysts then enter another person through the mouth and restart the cycle.

Among people living in areas where little attention is given to environmental sanitation, the carrier (better called the cyst-passer) undoubtedly plays an important part in spreading amebiasis. Ivanhoe [115] studied the presence of *E. histolytica* cysts in a children's home in New Orleans (U.S.) and was able to recover them from six places outside the body. This suggests that direct contact transfer aided by general pollution of the environment served to maintain a focus of infection in this home. Cleaning the home throughout with live steam and at the same time carrying out mass chemotherapy against *E. histolytica* on the home's attendants and its children proved to be a practical means of controlling amebiasis. If the same dirty conditions were permitted in the individual family home, amebiasis could readily spread among its occupants also. Although food handlers appear to be in a position to be important transmitters, Sapero [116] concluded that there was little evidence to support this belief. He based his judgment on a controlled investigation of 14 groups, including 919 persons, which had been served food by carriers of *E. histolytica.*

It has been shown that filth flies, including *Musca domestica,* have the ability to transmit cysts internally. Roberts [117] determined that, given access to infected material, the common fly will transmit *E. histolytica* cysts in its feces for a period up to at least 31 hours after the initial feeding and for nine hours the cysts will appear in its vomit-drops. By experiment, Pipkin [118] learned that the housefly was relatively unimportant as a cyst transmitter by external carriage.

In some countries, farm crops are fertilized with human excreta. Where this is done, eating raw leafy vegetables presents a real health threat. Also, washing vegetables to be eaten without thorough cooking, in water polluted with cysts provides an opportunity for the consumer to contract an amebic infection.

In 1925, Manson-Bahr [119] implicated water as a transmitter of amebiasis. On the Cape Verde Islands, he treated ten cases of this disease among persons whose sole source of infection, common to all, lay in water brought from Lisbon and stored in large tanks liable to contamination.

There are ample opportunities for amebic cysts deposited on the ground to be washed into small, unprotected water supplies and, no doubt, this takes place in some areas of the world resulting in sporadic infections and clinical cases which do not receive epidemiological or other confirmation. But recorded instances of water-borne epidemics are rare.

In the fall of 1933, two neighboring downtown hotels in Chicago, having in part a common water supply, were chiefly involved in an epidemic of amebiasis which caused at least 1,409 cases, two thirds of which were visitors

to that city from 400 other cities in the United States, Hawaii and Canada.[120] The two major points of possible pollution resulting in the water-borne infections in the hotels were (1) two cross-connections which joined an overhead sewer to condenser-water discharge pipes and (2) a sewer over a cooled drinking-water tank containing an old, rotting wooden plug which permitted leakage into the drinking-water tank. Another outbreak, smaller than the one in Chicago, but involving 'ne cases in 1950-1952 and 22 cases in the first five months of 1953 occurred at an industrial plant in Indiana (U.S.). Fassnacht[121] relates that here a six-inch water suction line passed through a sewer manhole and when this sewer became surcharged, a joint in the water line would be under a foot of sewage. By dye tests, the water-line joint was found to be leaking revealing the point of entrance of infected sewage.

The susceptibility of man to amebiasis is general. Many people harbor the infectious agent—the mature cyst—without showing clinical symptoms. There is no proof that man acquires any immunity as a result of infection.

The most logical measure to apply to prevent the communication of amebiasis is the sanitary disposal of human feces. If infected feces were not scattered about, people would not be subjected to the chance of eating the mature cysts. However, this way of preventing this disease is not wholly practicable in the world today; currently it is only an objective. Therefore, preventive action must include teaching personal hygiene to maintain clean fingers; cooking foods sufficiently to kill entrained cysts and avoiding eating those raw foods not free of cysts; excluding flies from living spaces and denying them access to foodstuffs; and providing safe drinking water at the point where the consumer receives it.

The cysts of *E. histolytica* are readily destroyed by drying. A temperature of 55 degrees C., well below boiling temperature, will also kill them. They will survive in water at about ten degrees C. for as long as a month. The work of Spector et al[122 123] has demonstrated that coagulation and rapid sand filtration provide an effective barrier to the ingress of cysts into water supplies. Cysts are resistant to the disinfectant, chlorine, in doses producing low residual chlorine concentrations. Fair et al,[124] experimentally working with 30 *E. histolytica* cysts per ml, accomplished their destruction with ammonia chloramines in 30 minutes at 23 degrees C. when the titratable chlorine residual in ppm was 2.0 at pH 4.0, 4.0 at about pH 7.6 and about 5.5 at pH 11.0. Free chlorine was about twice as effective as chloramines at pH ranges below 8.0 but was less effective at pH 11.0. Thus, superchlorination followed by dechlorination should safeguard water supplies if the distribution system is not invaded by infected sewage.

Chang and Morris[125] studied elemental iodine as an emergency disinfectant for drinking water and they concluded that a dilute aqueous iodine concentration of 5.0 to 10.0 ppm was effective against amebic cysts and other types of water-borne pathogenic organisms within 10 minutes at room temperature. "High germicidal activity is maintained over the pH range 3.0 to 8.0 in the presence of a variety of water contaminants. * * * Almost all waters can be made safe for drinking with a single dose of iodine of approximately 8.0 ppm."

INFECTIOUS HEPATITIS. Infectious hepatitis, as distinguished from serum hepatitis, is an acute infection, usually benign in character. In epidemics the case mortality rate is estimated to be less than 0.2 per cent.[126] The infectious agent is a virus. Some authorities believe that a more definitive way to discuss these two closely related infections would be to class both infectious hepatitis and serum hepatitis as viral hepatitis and ascribe infectious hepatitis to hepatitis virus A and serum hepatitus to hepatitis virus B.

Epidemic or infectious hepatitis has been known for thousands of years. Hippocrates, in the 5th century B.C., mentioned it in his writings, as did Pope Zacharias in 75 A.D. The modern concept of the virus etiology of the disease is largely the result of broad studies made during World War II when there were 170,000 cases reported in the U.S. Army alone.[60] Cameron [127] proved the presence of the infective agent in the blood in 1940-1941, and MacCallum and Bradley [128] found it in stools in 1944.

This disease is present throughout the world and is endemic in some areas. Usually it occurs in widespread outbreaks in rural and urban areas over a time-period of two to four months but sharp, localized epidemics do take place in institutions, among military groups and families, and under other circumstances.

Accurate data regarding the incidence of infectious hepatitis are not available but evidence indicates that both virus A and virus B are widely distributed. Imperfect estimates, based upon a World Health Organization report [39] of cases of infectious hepatitis notified to health authorities, added to those among inpatients and to those among outpatients of hospitals or dispensaries, for 1957 are as follows:

Africa	11,543
America	20,648[a]
Asia	6,694
Europe	151,096
Oceania	7,101
Total	197,082

[a] Including serum hepatitis in Alaska, Puerto Rico and United States

The reservoir of this disease is man and the sources of infection are feces and blood from infected persons. Most available evidence points to the infection of man through the mouth by the consumption of infected water, food including shellfish and perhaps milk, all vehicles of transmission susceptible to contamination by infected feces. The respiratory mode of spread has not yet been proven but present evidence clearly involves the person-to-person route in this disease's dissemination. Virus A is present in the blood during the incubation and acute illness phases in man and it can be spread then by contaminated medical instruments, blood and plasma. The role of carriers is not clear but it is known that chronic carriers do exist [126] who pass the virus in feces.

Polluted shellfish have been shown, by epidemiological procedures, to have been the vehicle of transmission of the virus of infectious hepatitis. Roos [129] implicated oysters (presumably eaten raw) in an epidemic of 119 cases in Goteberg, Sweden. Rindge [130] described a 1961 outbreak of this disease in Pascagoula, Mississippi (U.S.) where there were 50 cases of

which 74 per cent gave a positive history of having eaten raw oysters within 15 to 50 days prior to the onset of the disease; he also discussed a study of 370 adult cases of infectious hepatitis in New Jersey (U.S.) early in the same year in which it was found that 54.3 per cent had a history of having eaten raw clams during the same incubation period.

Murphy [131] studied a small milk-borne outbreak of ten cases of infectious hepatitis in Forsyth, Georgia (U.S.) in 1945. Of the eight households involved, seven were supplied with milk from the same dairy. Within the incubation period, cases appeared among consumers of milk from this dairy but not among non-consumers who were 20 times more numerous. The source of the infection was not discovered but it was known that there were two cases in close proximity to this dairy which was devoid of all sanitary conveniences for handling milk. In 1944, at a university in Ohio (U.S.), there were 24 cases of infectious hepatitis within 11 days among members of one fraternity and primarily among those members who ate regularly at the fraternity table. Read [132] studied this outbreak carefully and he could neither discover a source of infection other than food nor trace the outbreak to any previous cases.

An epidemic following the usual pattern of contact-spread, with 152 cases over the first six months of 1951, was investigated by Knight [133] and his associates. These cases were chiefly in school children in two adjacent rural school districts in Cooper County, Missouri (U.S.). Transmission could not be traced to any cause other than personal contact. The schools appeared to be the main centers of dissemination to families which when infected became foci. Another incident in which personal association between infected humans and susceptible ones was the important factor took place in 1944-1945 [134] in and near a small town in Iowa (U.S.). Of the 276 known cases of infectious hepatitis, 222 were investigated. The data indicated that water, milk or food was not involved and that spread must have been on a person-to-person basis.

In the absence of means to demonstrate the virus of infectious hepatitis other than by human volunteers, epidemiological researches into outbreaks occasionally lead the investigator to conclude that its spread has been due to more than one factor. Mosley [135] reached this viewpoint when he studied an 18-case outbreak in Daviess County, Kentucky (U.S.) in 1956. He hypothesized, with the aid of fluorescein dye tests, that half the cases resulted from the contamination of several private water supplies by the effluent from the septic tank used by the first case of the group, and that the remaining cases were due to contact.

The transmission of this disease by water is confirmed by many epidemiological incidents. In 1930-1931 in New Brunswick, Canada, there was an outbreak of 173 cases at three educational institutions. The epidemiological evidence compiled by Fraser [136] pointed to one of three water supplies as the vehicle of spread. Temporarily the town water supply was not suitable to use and recourse was had to an unprotected spring obviously badly contaminated. This source of water was blamed for the many infections.

An apparent water-borne outbreak reported by Neefe [137] occurred in 1944 in a Pennsylvania summer camp. Over a 13-week period, 350 of the 572 in

the camp were involved. The evidence implicated the well at the girls' camp which turned out to have cesspools on four sides of it, the closest being but 15 feet away. There was no outward proof that the cesspools had caused the contamination of the water but the rock formations were cracked and fissured shale and limestone, and underground percolation could readily have been taking place. Neefe believes this study to be the first one supplying satisfactory evidence of natural transmission of a viral agent by water.

In a Pennsylvania (U.S.) industrial city having a water purification plant including coagulation, filtration and chlorination, there were 52 cases of infectious hepatitis between October 1944 and April 1945. Hayward [138] found the only factor common to the cases to be the public water supply, the source of which was not infrequently contaminated with raw sewage. This outbreak was not an explosive one and this could be due to partial safeguarding of the public drinking water by the purification processes employed.

An outbreak of infectious hepatitis occurring in Delhi, India, during a six-week period starting the first of December 1955, was described by Viswanathan [139] "as an unprecedented epidemic of an explosive nature." There were 7,000 cases of manifest jaundice reported to the health authorities during the course of the epidemic but based upon an intensive sample survey the total case incidence was estimated to be 97,600 and the case mortality rate 0.9 per 1,000. The main drinking water pumping station supplying 90 per cent of the urban population of the city draws its raw water from the Jumna River. About 700 feet downstream from the intake, a drainage ditch discharged large quantities of raw sewage. Due to low water in the river and the suction effect of the water pumps, it is estimated that about the middle of November, 50 per cent of the water plant raw water pumpage was from the drainage ditch. Massive infection of the city's water supply took place during four days. Intensification of the filtration and disinfection processes did not inactivate the infectious virus though it may have produced some attenuation of it.

Susceptibility to infectious hepatitis is variable although the disease appears to have a predilection for school-age children, particularly those under 15 years. This susceptibility is far from a constant one; for example, in the Delhi outbreak, the highest case rate was in the 15-34 age group and this rate was far higher than that for the group under 15 years. Epidemiological data suggest that an infection with hepatitis virus A is followed by resistance to infection with the homologous virus and it is believed that the duration of immunity to the A virus may be long-lived.[120]

The sanitary disposal of feces to keep infected excreta from reaching a vehicle of transmission and the exercise of good habits of personal cleanliness would go far to reduce the incidence of this disease. They would not, however, be adequate to cut the disease's spread entirely because of the obvious part played by contact and person-to-person dissemination. The virus of infectious hepatitis is very hardy; it will survive heating to 56 degrees C. for at least 30 minutes and it is not killed by freezing, drying or various preservatives.

Neefe,[140] after his 1947 studies, concluded that coagulation, settling and filtration with a diatomite filter of contaminated water was not effective because 40 per cent of the volunteers who ingested the treated water developed the disease. The purification process did prolong the incubation period and reduce the incidence 40 per cent. When the treated water was disinfected with chlorine in sufficient quantity to provide, after 30 minutes contact, total and free residual chlorine concentrations of 1.1 and 0.4 ppm respectively, the virus was inactivated. Chlorinating the contaminated water prior to the purification process was not effective against the virus.

DIARRHEAL DISEASES. Diarrhea means the frequent passage of loose or fluid stools; it may be the primary symptom or it may be secondary in other diseases having a definite etiological cause.

Messih [141] has arranged diarrhea and the agents or factors responsible for it in three classes, as follows:

1. Primary diarrhea resulting from infections
2. Secondary diarrhea appearing as a symptom accompanying an infection outside the intestines and due to toxemia produced by the etiological agent of the particular disease
3. Non-infectious diarrhea caused by:
   a. Chemicals, as in food poisoning
   b. Gastrointestinal allergies
   c. Metabolic disorders like those associated with emotional excitement
   d. Irritation of the intestinal tract such as that which can result from the consumption of foodstuffs containing a large proportion of indigestible material

Primary or infectious diarrhea is related to a variety of infectious agents, some of which have been confirmed and some of which are still only suspected. Among these are:

1. Bacteria
   a. Cholera vibrio (See Cholera)
   b. *Shigellae* (See Bacillary Dysentery)
   c. *Salmonellae*, the infections from which may be evident as
      (1) Paratyphoid fever (See Paratyphoid Fever)
      (2) Typhoid fever (See Typhoid Fever)
      (3) Gastroenteritis due to contaminated food
      (4) Asymptomatic salmonellosis
   d. *Staphylococcus aureus*. Infections with this organism may result in
      (1) Food intoxication of bacterial origin
      (2) Post-operative enteritis
      (3) Infantile gastroenteritis
   e. *Clostridium perfringens*, another organism having the capability of producing a toxin giving rise to food poisoning [142]
   f. *Escherichia coli* of certain types, several of which have been found to be connected with sporadic and epidemic diarrheal disease in infants
   g. Doubtful pathogens such as *Paracolobactrum arizonae*, which causes diarrheal syndromes similar to those due to *Salmonellae*, *Proteus morganii*, *Proteus vulgaris* and *Pseudomonas aeruginosa*, all held responsible for some "summer diarrhea"
2. Helminths, illustrated by the schistosomes and nematodes (See Dracontiasis and Schistosomiasis)
3. Protozoa such as *Entamoeba histolytica* (See Amebiasis)

4. Viruses. Some of the ECHO (Enteric Cytopathogenic Human Orphans) viruses have been incriminated.
5. Fungi. The intestinal type of candidiasis (moniliasis) is an example of this variety of diarrhea-producing infection.

The most important of the diarrheal diseases, from the public health standpoint, are those due to the *Shigellae*, the *Salmonellae*, the *E. coli*, and the parasites. These cause the largest proportion of the disability associated with diarrhea.

Distribution of morbidity and mortality from diarrheal diseases varies from country to country but it seems clearly evident that the rates of illness and death are closely related to the level of the environmental sanitation conditions, personal habits of cleanliness, the ability to have and maintain a proper diet, and the socio-economic and cultural standards. When these aspects of living are primitive, the diarrheal diseases flourish. Age distribution differs according to the causative agent and from country to country. Broadly speaking, *E. coli* and ECHO viruses infections strike hardest at infants under one year of age. Children aged one to four years are most susceptible to infection by the *Shigellae* and *Salmonellae* although it may occur in persons of any age. Parasite infections are most numerous in the older age groups.

Sound data on illness caused by these infectious agents can be obtained only through painstaking epidemiological studies on a community basis. The accuracy of mortality figures is determined by the accuracy of the original diagnosis and diagnoses of diarrheal disease are chiefly symptomatic. Confused and confusing terminology applied both to the etiologic agents and the disorders, the different bases used for the classification of diseases, failure to give any medical diagnosis on death certificates and failure to record many infant deaths lead to the belief that the magnitude of the diarrheal disease problem could be seriously underestimated by relying only on the mortality figures now available.

The difficulty of supporting accurate data on deaths from diarrheal diseases with reliable diagnoses is illustrated by the findings of Zobel [143] in his study in 1956 of the use of a revised death certificate in an experimental area in two districts, including both urban and rural territory, in the state of Lara, Venezuela. Here the main "cause of death * * * was enteritis, including diarrhea and dysentery." Excluding those caused by accidents and violence, enteritis was responsible for 18.2 per cent of the deaths, and of these a medical diagnosis was made in 16.8 per cent prior to death and in 21.2 per cent subsequent to death. In the latter cases, the cause of death was certified to by the physician based upon the observations of others or by the civil authority.

No area of the world is free from diarrheal diseases. It is impractical to bring together all available data measuring illnesses and deaths resulting from all the diarrheal diseases but some data are shown in tables 1, 2, 3, 4, 6, and 7 and these have their shortcomings. Fairly representative of the death rates attributed to some of the diarrheal diseases are the data shown in table 8 which pertain only to countries in North and South America. The

body disturbances included in this tabulation are those with title numbers 543, 571 and 572 in the International Statistical Classification of Diseases, Injuries and Causes of Death [144] and are defined as follows:

543 — gastritis and duodenitis
571 — gastroenteritis and colitis, except ulcerative; age four weeks and over
572 — chronic enteritis and ulcerative colitis, excluding amebiasis

It is unlikely that the death rates due to these diseases will be very different in those countries not included in this table but comparable in their state of development.

TABLE 8

Number of Deaths and Death Rates per 100,000 Population from Gastritis, Enteritis, etc., in the Americas, 1956 *

Country (For Footnotes, see TABLE 4)	Number of Deaths	Death Rate per 100,000 Population
Argentina [a]	3,014	16.4
Bolivia [a]	1,771	56.7
Brazil [b]	9,421	157.1
Canada [c]	905	5.6
Chile	5,645	85.7
Colombia	15,638	120.9
Costa Rica	1,221	123.6
Dominican Republic [d h]	2,645	104.9
Ecuador [d e]	3,928	106.4
El Salvador [d]	4,951	225.8
Guatemala [d]	8,808	270.3
Honduras [f]	640	39.2
Mexico [d]	67,563	227.6
Nicaragua	1,191	92.5
Panama	490	55.3
Paraguay [g]	264	16.5
Peru [a]	3,883	48.6
United States [d]	7,754	4.7
Uruguay [d]	524	20.0
Venezuela	5,577	96.6

* See reference (19). (Adapted from TABLES 5 and 6 thereof)

The relation of diarrheal disease to the death of infants under one year and of young children in the one to four year age group is so important as to merit special attention. The study of diarrheal disease in the Americas by Verhoestraete and Puffer [145] emphasizes that diarrheal disease is a major health problem in those countries which are still under-developed economically. Conversely, in areas which have improved in economic stature, progress has been made since the beginning of the century in reducing death rates from this affliction. There are many difficulties in attempting to arrive

at comparable mortality figures for these two age groups; in the Americas, the classification of diseases and terminology are two deterrents to this end. In ten selected countries of the Americas, during the period in which the study was made, the disease group labeled "diseases of early infancy" ranked first as the cause of infant mortality in eight; in the other two (Venezuela and the state capitals of Brazil), diarrheal disease was the leading cause. In eight of these ten countries (excluding Canada and the United States), the death rates for infants from diarrheal disease were 700 or more per 100,000 live births. Among children one to four years old, diarrheal disease is the most important cause of death in six of the ten selected countries.

The pathogenic agents of the diarrheal diseases are almost without exception transmitted by contact with an infected person, either directly or indirectly. There are many vehicles which assist in the dissemination of these diseases; water, foods, shellfish, fish, ice, milk, milk products, raw fruits and vegetables, and flies all perform this undesirable service. Susceptibility of man to diarrhea varies as does the degree of immunity acquired following an infection.

The two major approaches to accomplishing a reduction of incidence of the diarrheal diseases are education and the betterment of the sanitary conditions of the environment. Health education can make a great impact on the problem by teaching personal hygiene, sanitation of and in the home, proper care of babies and pregnant women, how to recognize the illness and the need for skilled management of it, and ways to improve diet.

With respect to making the environment less hazardous, emphasis should be on the vehicles of transmission to cut the lines of spread. In connection with the continuous efforts to make safe drinking water available to all, it should be pointed out that this is not likely to produce as dramatic a reduction of incidence of diarrheal disease as it did in the case of typhoid fever. For example, at the close of the period 1900-1920, there were 12 million people in 16 selected cities in the United States using filtered and chlorinated drinking water. This improvement in water quality was an important influence on the 91.0 per cent reduction of the typhoid fever death rate in this population group. However, during the same period, the death rate due to diarrhea and enteritis in this same group declined only 44.0 per cent.[145] As mentioned heretofore, the ready availability of safe water both to drink and for self-cleansing purposes will reduce infections by *Shigellae,* a most important item in this array of diarrhea-causers. Verhoestraete[145] has drawn attention to the threat of animals infected with *Salmonellae,* other than *S. typhi,* because of the close relation between man and animals in primitive homes and their surroundings.

There is much information available regarding methods of preventing the diarrheal diseases. While this knowledge is being used in practical control programs, epidemiological investigations of causative agents and the factors of the agent-host-environment relationship should be pressed forward to unfold new and better procedures upon which to build more effective and economical control efforts.

PLEURODYNIA. Pleurodynia is an acute infectious disease usually occurring in localized epidemic form among children and young adults. According to Harrison [60] it is never fatal. The infectious agents are various viruses of the Coxsackie Group B.

Epidemic outbreaks have been reported in Australia, Europe, England, New Zealand and North America. Huebner [146] and his associates investigated an outbreak of 22 cases in 1951 in northeastern Texas and were able to recover Coxsackie Group B virus from the stools of 16 of those persons exhibiting signs and symptoms of the disease.

Man is the reservoir of the infective viruses which appear in the feces and throat discharges of those having the disease. No animal is known to serve as a reservoir although Coxsackie viruses have been found on flies and mosquitoes, and Melnick [147] states that they persist in flies and their excreta for one to two weeks.

The method of spread of pleurodynia is not clear but the most likely hypothesis is that transmission is by way of contact with an infected person or through intermediate articles such as utensils or towels which have been freshly soiled with infective material.

In 1954 and 1956, Kelly [148] examined samples of sewage at the Albany, New York (U.S.) sewage treatment plant to ascertain if enteric viruses were present in it. Of 20 samples taken in 1954, Coxsackie Group B virus was isolated from eight; in 1956, the number of samples containing this virus was found to be 12 of 17. Isolations were made from samples of raw sewage and treatment plant effluent, and from samples taken at the plant outfall and at a point 25 feet downstream from the point of outfall discharge. Melnick [147] examined urban sewage and flies in six states over a period of four years and in doing so made 1,929 tests. Of these, 269 yielded Coxsackie virus but all isolations were members of Group A. It was found that this type of virus was generally present in sewage in the summer and fall but absent in the winter and spring; it was found both in the influent and effluent at sewage disposal plants.

After performing certain experimental studies on the ability of sewage treatment plants, without effective chlorination, to inactivate Coxsackie virus, Kelly [149] concluded that there was "ample evidence that sewage treatment, while complying with existing standards, may not control pathogenic agents to the extent that receiving streams can be ignored as potential sources of infection."

It is evident that Coxsackie viruses are chiefly feces-borne and that they are readily isolated from sewage and sewage effluents. This being the case, they must be considered as potential water-borne viruses which may be present in raw waters drawn from water courses for use, following purification, as domestic water supplies. Coxsackie viruses are relatively stable. Clarke [150] found that the time required to reduce the number of Group A Coxsackie viruses 99.9 per cent in Ohio River (U.S.) water at a temperature of 20 degrees C. was approximately six days and at a temperature of 8 degrees C.,

12 to 16 days. This infective agent is, therefore, likely to be present in flowing sources of raw water which are located not too distant from upstream points of sewage discharge.

As a safeguard against infection by Coxsackie virus via public water systems, an effective water purification process is essential. Chang et al [151] determined, in experimental work employing Group A Coxsackie virus, that the flocculation process satisfactorily applied would remove 95-99 per cent of the organisms. In other work, using a purified suspension of Group A Coxsackie virus, Clarke [152] found that 99.6 per cent inactivation was reached in three minutes at pH 7.0 and temperature of 27-29 degrees C. when the residual of free chlorine was 0.5 ppm. The inactivation time was dependent upon temperature, pH and chlorine concentration.

In spite of the known presence of Group B Coxsackie virus in raw waters available to water purification plants, *there is no epidemiological evidence to prove that water is a vehicle of transmission of pleurodynia.*

"Susceptibility [to this disease] is probably general, and presumably a type-specific immunity results from infection." [5]

Rivers [153] states that "specific measures to control infection by Coxsackie viruses have not been devised."

POLIOMYELITIS. Poliomyelitis is an acute disease which, in its most characteristic form, involves parts of the central nervous system. In most of the patients, however, the illness is mild and is limited to upper respiratory or gastrointestinal symptoms. Fatality in mild attacks varies from 2 to 10 per cent; in the gravest form, bulbar poliomyelitis, fatality ranges from 5-60 per cent. The infectious agents are poliovirus, types 1, 2, and 3.

This disease is world-wide in its distribution. It occurs both sporadically and in epidemics. Incidence seems to be highest in the summer and early fall but this varies from year to year and from region to region. Children from 1 to 16 years of age are more frequently attacked than adults but in several areas this pattern is changing to the disadvantage of older children and young adults. In countries where artificial immunization has been widely practiced, epidemics have been confined largely to the least vaccinated population groups.

The reservoir of the infective polioviruses is man, most frequently children suffering from clinically unrecognized or inapparent infections. The sources of the infection are the pharyngeal secretions and the feces of infected persons. Harrison [60] states that "the greatest quantity of virus is found in the small intestine."

Currently there is almost uniform agreement that the virus of poliomyelitis is spread principally by person-to-person contact and the portal of entry is the mouth. The bulk of the evidence revealed by epidemiological research related to this disease supports this conclusion. However, milk has served, in a few instances, as a transmitting vehicle and drinking water is still suspect.

There is also some evidence that flies may play a part in spreading this disease.

In 1926, in Broadstairs, England, an epidemic of poliomyelitis having unusual localizing features and involving 75 cases was studied by Aycock.[154] Among nine sources of milk, that from two dairies (A and F) appeared to be responsible for 69 of the cases. Dairy A may even have had a greater responsibility because it sold to Dairy F during the epidemic period, two-thirds of the milk Dairy F distributed. The evidence pointed to contamination of only one grade of Dairy A's milk, coming from a single farm, as the source of infection of these cases.

Eight cases of this disease (two more cases were subsequently reported) occurring in 1925 in Cortland, New York (U.S.) were studied carefully by Knapp and his associates.[155] These few cases, in a city of 15,000 people, were subsequent to the appearance of three other cases more than two months before. With the possible exception of one, all of these cases were people who drank milk from one dealer whose sales constituted only 4 per cent of the city's total milk supply. These cases were dispersed widely enough to make it improbable for the milk supply to be a mere coincidence of geographic distribution. A case of paralytic poliomyelitis existed on the farm from which most of the milk came, and this patient, although showing signs of illness, was milking cows and otherwise handling the milk for four days just prior to the onset of paralysis.

During the summer of 1941, Trask and Paul[156] collected 19 samples of flies in epidemic areas both during and after poliomeylitis epidemics. Of eight samples obtained during the latter part of a local epidemic but within ten days of the onset of a local case of poliomyelitis, four yielded poliovirus. In four cases, potential sources of virus (feces) of recent origin were available within a few yards of the points where the flies were captured; three of these yielded poliovirus. According to Melnick,[157] poliovirus can be recovered from two genera of blow flies and the common housefly, *Musca domestica,* and their excreta collected over a period of several days following their feeding on infected human excreta.

The suspicion attached to drinking water as a vehicle of spread of poliomyelitis arises from the fact that the infectious agents have been found in sewage. In 1941, Paul and Trask[158] stated that "it has become increasingly apparent that the virus of poliomyelitis may be readily isolated from stools of patients with poliomyelitis." An examination of sewage collected regularly for about 12 months at two sewage treatment plants in Toronto, Canada, resulted in the recovery of poliovirus by Rhodes et al[159] at the height of an epidemic in July-September, 1959. Virus was isolated at one of the plants five weeks before the first reported case of poliomyelitis in the district contributing sewage to that plant. In 1954, Kelly[148] examined 20 samples of sewage collected at the Albany, New York (U.S.) sewage treatment plant and isolated poliovirus from 13 of them; in 1956, of 17 samples studied, poliovirus was found in three.

The presence of the polioviruses in sewage assures its presence in some raw water supplies which may or may not be drawn upon as sources of

domestic water. As May [6] has so aptly put it in discussing disease spread in the United States, "since there is a general practice of discharging sewage into rivers and streams, there exists a great proportion of citizens who, in whatever purified form, drink each other's sewage." Where available, sewage purification works interpose a barrier against the pollution of raw water with poliovirus but neither are such works everywhere available nor are those in use always entirely effective in producing a biologically safe effluent. The virus is quite resistant and will live in water and sewage for as long as four months.[60]

Water purification systems constitute a second defense against contamination of domestic water supplies by polioviruses which may be present in raw waters. Although the best measure to employ to avoid the possibility that municipal water supplies will contain poliovirus is to shun the use of grossly polluted water as a source of supply, communities have not always been able to do this. Chang [160] has suggested that such water should be prechlorinated at the rate which will produce 0.3-0.4 ppm of free residual chlorine during the entire treatment process and 0.2-0.3 ppm at the time the water leaves the plant. He adds, also, that "if the water treatment process includes satisfactory flocculation, another 90-99 per cent virus removal will be added to the total efficiency."

The part that drinking water may play in the transmission of poliomyelitis was discussed by Krumbiegel [161] in 1944 and he concluded that the epidemiological evidence then available failed to show that water was biologically of any importance as a transmitter of poliovirus. Similarly, in 1949, Maxcy [162] summarized current opinion to the effect that epidemiological studies of this disease had not resulted in proof that the pollution of a community water supply was responsible for the exposure of consumers to infection with poliovirus.

Within the last few years, two poliomyelitis outbreaks have been studied and drinking water has been suggested as the vehicle of transmission in both instances. Little [163] believed the Edmonton, Canada, episode in 1953 to be water-borne because the outbreak was an explosive one, with scattered cases within the city; it occurred at the time when there were lapses in the chlorination of the settled sewage of Devon, which reached the North Saskatchewan River, the source of Edmonton's raw water; and poliovirus was recovered from the Devon sewage. In the Huskerville (Lincoln), Nebraska (U.S.) situation, described by Bancroft,[164] an abrupt, severe outbreak occurred in an unusually homogeneous community under circumstances permitting an analysis of possible etiological factors. During a five-week period, more than 10 per cent of 347 children in the area of high incidence developed poliomyelitis while there were no cases of the disease—either non-paralytic or paralytic—among 256 children living in the adjoining section of the village. There was found to be a close correspondence between the location of cases and of flush-valve water closets not provided with vacuum breakers, as well as a correspondence in time between the outbreak and the occurrence of extreme fluctuations of pressure in the water

mains.* Immunological studies corroborated the belief that poliovirus, type 1, had been carried by contaminated drinking water.

In view of the paucity of information bearing upon *water as a vehicle of spread of poliomyelitis, its conviction as such will have to wait until there is a better understanding of the disease process of poliovirus and the epidemiology of the disease has been more thoroughly investigated.*

Man is generally susceptible to this disease and type-specific resistance of long duration follows both apparent and inapparent infections.

Active immunization is presently the best preventive measure.

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* In an Editor's Note following reference (160), it is stated that "water department pressure records contradict the supposition that back siphonage could have occurred."

REFERENCES

5. Control of Communicable Diseases in Man. American Public Health Association, New York (9th ed., 1960).

6. May, J. M. The Ecology of Human Disease. MD Publications, Inc., New York (1958).

7. Annual Epidemiological and Vital Statistics, 1956. World Health Organization, Geneva (1959).

8. Weekly Epidemiological Record, No. 3 (1960). World Health Organization, Geneva.

9. Snow, John. Snow on Cholera. The Commonwealth Fund, New York (1936). (A reprint of Snow's paper "On the Mode of Communication of Cholera"—1854).

10. Pollitzer, R. Cholera. Monograph Series No. 43. World Health Organization, Geneva (1959).

11. Lara, H. Interesting Features of a Rural Outbreak of Cholera Due to Infected Drinking Water. Am. J. Hyg., 7:606 (1927).

12. Read, W. D. B. and Pandit, S. R. Distribution of V. Cholerae and El Tor Type Strains in Certain Rural Areas in India. Ind. J. Med. Res., 29:403 (1941).

13. Maxcy, K. F. (Editor) Rosenau's Preventive Medicine and Hygiene. Appleton-Century-Crofts, Inc., New York (8th ed., 1956).

14. Robertson, R. C. and Pollitzer, R. Cholera in Central China During 1938—Its Epidemiology and Control. Tr. Roy. Soc. Trop. Med. & Hyg., 33:213 (1939).

15. Pollitzer, R. A Note on the Incidence and Epidemiological Importance of Cholera Carriers. Bulletin 7:359 (1952). World Health Organization, Geneva.

16. Official Records of the World Health Organization, No. 18, p. 110, Geneva (1949).

17. Dubos, R. J. (Editor) Bacterial and Mycotic Infections of Man. J. B. Lippincott Co., Philadelphia (3rd ed., 1958).

18. Demographic Yearbook, 10th issue, 1958. United Nations, New York.

19. Summary of Four-year Reports on Health Conditions in the Americas. Scientific Publication No. 40 (1958). Pan American Sanitary Bureau, Washington. (Adapted from TABLE 6 thereof.)

20. Caldwell, W. A. and Hardwick, S. W. Observations on Flexner Dysentery. Edinburgh Med. J., 50:129 (1943).

21. Chesley, F. F. and Woolsey, C. I. Frequency of Bacillary Dysentery at the Cook County Hospital, Chicago, Ill. Gastroenterology, 2:258 (1944).

22. McGinnes, G. F., McLean, A. L., Spindle, F. and Maxcy K. F. A Study of Diarrhea and Dysentery in Henrico County, Virginia. Am. J. Hyg., 24:552 (1936).

23. Nisbet, B. R. Report on Outbreak of Flexner Dysentery. The Med. Officer, 72:133 (1944).

24. Kuhns, D. M. and Anderson T. G. A Fly-borne Bacillary Dysentery Epidemic in a Large Military Organization. Am. J. Pub. Health, 34:750 (1944).

25. Godfrey, E. J. and Pond, M. A. A Hospital Epidemic of Flexner Dysentery Caused by Contaminated Ice. J.A.M.A., 114:1151 (1940).

26. Auché, M. A. Transport des Bacilles Dysentériques para les Mouches. C. R. Soc. Biol. (Paris), 61:405 (1906).

27. Orton, S. T. and Dodd, W. L. Experiments on Transmission of Bacteria by Flies, with Special Reference to an Epidemic of Bacillary Dysentery at the Worcester State Hospital, Massachusetts. Boston Med. & Surg. J., 163:863 (1910).

28. Cox, C. R. Water-borne Gastro-enteritis. Jour. AWWA, 31:1489 (1939).

29. Eliassen, R. and Cummings, R. H. Analysis of Water-borne Outbreaks, 1938-1945. Jour. AWWA, 40:509 (1948).

30. Wolman, A. and Gorman, A. E. The Significance of Water-borne Typhoid Fever Outbreaks, 1920-1930. Jour. AWWA. 23:160 (1931).

References (continued)

31. Sjoblom, M. C. The Outbreak of Typhoid and Dysentery at the Chicago and Alton Shops in Bloomington. Jour. AWWA, 7:560 (1920).

32. Kinnaman, C. H. and Beelman, F. C. An Epidemic of 3,000 Cases of Bacillary Dysentery Involving a War Industry and Members of the Armed Forces. Am. J. Pub. Health, 34:948 (1944).

33. Freitag, J. L. A Water-borne Outbreak of Dysentery. Health News, 37:4:4 (1960).

34. Watt, J., Hollister, A. C., Jr., Beck, M. D. and Hemphill, E. C. Diarrheal Diseases in Fresno County, California. Am. J. Pub. Health, 43:728 (1953).

35. Stewart, W. H., McCabe, L. J., Jr., Hemphill, E. C. and DeCapito, T. The Relationship of Certain Environmental Factors to the Prevalence of Shigella Infection. Am. J. Trop. Med., 4:178 (1955).

36. Hollister, A. C., Jr., Beck, M. D., Gittlesohn, A. M. and Hemphill, E. C. Influence of Water Availability on Shigella Prevalence in Children of Farm Labor Families. Am. J. Pub. Health, 45:354 (1955).

37. Gauld, R. L., Crouch, W. L., Kaminsky, A. L., Hullinghorst R. L., Gochenour, W. S., Jr., and Yager, R. H. Leptospiral Meningitis—Report of Outbreak Among American Troops on Okinawa. J.A.M.A., 149:228 (1952).

38. Yager, R. H. and Gochenour, W. S., Jr. Leptospirosis in North America. Am. J. Trop. Med. & Hyg., 1:457 (1952).

39. Annual Epidemiological and Vital Statistics, 1957. World Health Organization, Geneva (1960).

40. Reported Cases of Notifiable Diseases in the Americas, 1949-1958. Scientific Publication No. 48. Pan American Health Organization, Washington (1960).

41. Doherty, R. L. and Emanuel, M. L. An Outbreak of Letptospirosis in North Queensland. Med. J. Australia, 1 (43rd year):276 (1956).

42. Ward, T. G. and Turner, T. B. Study of Certain Epidemiological Features of Leptospiral Jaundice in Baltimore. Am J. Hyg., 35:122 (1942).

43. Ivy, J. H., Billings, E. R. and Elliott, T. A. Human Infection with *Leptospirosis Pomona* in Indiana. J. Indiana Med. A., 48:1095 (1955).

44. Van der Hoeden, J. The Epidemiology and Epizootiology of Leptospirosis in Israel. J. Trop. Med. & Hyg., 58:202 (1955).

45. Williams, H. R., Murphy, W. J., McGroan, J. E., Starr, L. E. and Ward, M. K. An Epidemic of Canicola Fever in Man with the Demonstration of *Leptospira Canicola* Infection in Dogs, Swine and Cattle. Am. J. Hyg., 64:46 (1956).

46. Schaeffer, M. Leptospiral Meningitis. J. Clin. Invest., 30:670 (1951).

47. Mikhailovskii, B. N. A Contribution to the Question of the Epidemiology of Anicteric Leptospirosis (Water Fever). J. Microbiol., Epidemiol. & Immunobiol. (USSR), 29:1444 (1948).

48. Chang, S. L., Buckingham, M. and Taylor, M. P. Studies on Leptospira Icterohaemorrhagiae. J. Inf. Dis., 82:256 (1948).

49. Kirschner, L. and Maguire, T. Survival of Leptospira Outside Their Hosts. N. Zealand Med. J., 56:385 (1957).

50. Taylor, E. W. The Examination of Waters and Water Supplies. J. and A. Churchill Ltd., London (7th ed., 1958).

51. Breed, R. S., Murray, E. G. D. and Smith, N.R. Bergey's Manual of Determinative Bacteriology. Williams & Wilkins Co., Baltimore (7th ed., 1957).

52. Saphra, I. and Winter, J. W. Clinical Manifestations of Salmonellosis in Man. N. England J. Med., 256:1128 (1957).

53. Rimpau, W. (Abstract). A Water-borne Typhoid Epidemic in Neuötting. Bull. Hyg., 26:23 (1951).

54. (Abstract). A Water-borne Outbreak of Paratyphoid B Fever. Bul. Hyg., 17:306 (1942).

References (continued)

55. Bardos, V. (Abstract). A Water-borne Epidemic of Paratyphoid A. Bull. Hyg., 28:793 (1953).

56. Parry, S. C. A Water-borne and Milk-borne Outbreak of Paratyphoid B Infection in Worcestershire. Brit. Med. J., II:661 (1942).

57. Kelman, J. Water-borne and Milk-borne Paratyphoid B. Health Bull. (Scotland), III:38 (1944).

58. McCoy, G. W. and Chapin, C. W. Studies of Plague, a Plague-like Disease, and Tuberculosis Among Rodents in California. U. S. Public Health Service Pub. Health Bull. No. 53 (1912).

59. Schmidt, B. Der Einbruch der Tularämie in Europa. (The Introduction of Tularemia in Europe.) Ztschr. f. Hyg. u. Infectionskr., 127:139 (1948).

60. Harrison, T. R., Adams, R. D., Beeson, P. B., Resnik, W. H., Thorn, G. W. and Wintrobe M. M. Principles of Internal Medicine. McGraw-Hill Book Co., Inc., New York (2nd ed., 1954).

61. Simons, S. A., Stevens, I. M. and Reeves, W. C. Some Epidemiological Observations on Tularemia in California 1927-1951. Am. J. Trop. Med., 2:483 (1953).

62. Hillman, C. C. and Morgan, M. T. Tularemia: Report of a Fulminant Epidemic Transmitted by the Deer Fly. J.A.M.A., 108:538 (1937).

63. Amoss, H. L. and Sprunt, D. H. Tularemia: Review of Literature of Cases Contracted by Ingestion of Rabbit * * *. J.A.M.A., 106:1078 (1936).

64. Francis, E. Tularemia. Am. J. Nursing, 34:1 (1934).

65. Karpoff, S. P. and Antonoff, M. I. The Spread of Tularemia Through Water, as a New Factor in its Epidemiology. J. Bact., 32:243 (1936).

66. Tsareva, M. I. (Abstract). A Water-borne Outbreak of Tularemia. Bull. Hyg., 21:307 (1946).

67. Falk, A. Water-borne Tularemia. Minnesota Med., 30:849 (1947).

68. Parker, R. R., Steinhaus, E. A., Kohls, G. M. and Jellison, W. L. Contamination of Natural Waters and Mud with *P. tularensis* and Tularemia in Beavers and Muskrats in the Northwestern United States. Nat. Inst. Health Bull. No. 193, Washington (1951).

69. Foote, H. B., Jellison, W. L., Steinhaus, E. A. and Kohls, G. M. Effect of Chlorination on *Pasteurella tularensis* in Aqueous Suspension. Jour. AWWA, 35:902 (1943).

70. Statistical Abstract of Ceylon, 1959. Government Press, Colombo, Ceylon.

71. Clark, B. M. Two Interesting South African Typhoid Outbreaks. South African M. J., 13:806 (1939).

72. Caraway, C. T. and Bruce, J. M. Typhoid Fever Epidemic Following a Wedding Reception. Pub. Health Rep., 76:427 (1961).

73. Lumsden, L. L., Hasseltine, H. E., Leake, J. P. and Veldee, M. V. A Typhoid Fever Epidemic Caused by Oyster-borne Infection (1924-1925). U. S. Public Health Service Supp. No. 50 to the Pub. Health Rep., (1925).

74. Ferguson, H. F. An Epidemic of Typhoid Fever Due to the Use of a Polluted Water Supply at the 1915 Assembly of Old Salem Chautauqua. Jour. AWWA, 3:874 (1916).

75. Neill, W. A., Martin, J. D., Belden, E. A. and Trotter, W. Y. A Widespread Epidemic of Typhoid Fever Traced to a Common Exposure. N. England J. Med., 259:667 (1958).

76. Wolman, A. (Editorial). Is Typhoid Fever a Vanishing Disease? Jour. AWWA, 18:497 (1927).

77. Waring, F. H. The Typhoid Fever Epidemic at Xenia, Ohio. Jour. AWWA, 6:167 (1919).

78. Horton, T. Typhoid Fever Outbreak at Tonawanda, New York. Jour. AWWA, 7:176 (1920).

References (continued)

79. Healy, W. A. and Grossman, R. P. Water-borne Typhoid Epidemic at Keene, New Hampshire. Jour. N. England Wat. Wks. Assoc., 75:38 (1961).

80. Beard, P. J. Longevity of Eberthella Typhosus in Various Soils. Am. J. Pub. Health, 30:1077 (1940).

81. Johnson, G. A. The Typhoid Toll. Jour. AWWA, 3:249 (1916).

82. Water Quality and Treatment. American Water Works Association, New York (2nd ed., 1951).

83. Bruns, H. Typhoid Fever Epidemics from Water Supply in Germany. Jour. AWWA, 25:1 (1933).

84. Morzycki, J. (Abstract). Investigations on Typhoid Carriers in Endemic and Epidemic Areas. Bull. Hyg., 25:15 (1950).

85. Butterfield, C. T. Bactericidal Properties of Chloramines and Free Chlorine in Water. Pub. Health Rep., 63:934 (1948).

86. Belding, D. L. Textbook of Clinical Parasitology. Appleton-Century-Crofts, Inc., New York (2nd ed., 1952).

87. Stoll, N. R. This Wormy World. J. Parasit., 33:1 (1947).

88. Gradwohl, R. B. H., Soto, L. B. and Felsenfeld, O. Clinical Tropical Medicine. The C. V. Mosby Co., St. Louis (1951).

89. Moorthy, V. N. An Epidemiological and Experimental Study of Dracontiasis in Chitaldrug District. Ind. Med. Gaz., 67:498 (1932).

90. Pradhan, Y. M. Observations on Experiments Designed to Combat Dracontiasis in an Endemic Area by Col. Morison's Method of Liming Wells. Ind. J. Med. Res., 18:443 (1930).

91. Ramakrishnan, N. R. and Rathnaswamy, G. K. Use of DDT for the Control of Cyclops Breeding and as an Anti-dracontiasis Measure. Ind. Med. Gaz., 88:386 (1953).

92. Moorthy, V. N. and Sweet, W. C. A Biological Method for the Control of Dracontiasis. Ind. Med. Gaz., 71:565 (1936).

93. Faust, E. C. Human Helminthology. Lea and Febiger, Philadelphia (2nd ed., 1939).

94. Heinz, H. J. and Brauns, W. The Ability of Flies to Transmit Ova of Echinococcus Granulosus to Human Foods. South African J. Med. Sci., 20:131 (1955).

95. Loucks, H. H. Hydatid Cyst. Nat. Med. Jour., China, 16:402 (1930).

96. Second African Conference on Bilharziasis. Technical Report Series, No. 204. World Health Organization, Geneva (1960).

97. Sullivan, R. R. and Ferguson, M. S. Studies on Schistosomiasis Japonica: III. An Epidemiological Study of Schistosomiasis Japonica. Am. J. Hyg., 44:324 (1946).

98. Hinman, E. H. Schistosomiasis—Characteristics and Problems of Control. Mimeographed paper. Office of Public Health, International Cooperation Administration, Washington (1960).

99. Jones, M. F., Newton, W. L., Weibel, S. F., Warren, H. B., Steinle, M. L. and Figgat, W. B. Studies of Schistosomiasis: The Effects of Sewage Treatment Processes on the Ova and Miracidia of Schistosoma Japonicum. Nat. Inst. Health Bull. No. 189, Washington (1947).

100. Jones, M. F. and Brady, F. J. Studies on Schistosomiasis: Effects of Water Treatment Processes on Schistosome Cercariae. Nat. Inst. Health Bull. No. 189, Washington (1947).

101. Witenberg, G. and Yofe, J. Investigation on the Purification of Water with Respect to Schistosome Cercariae. Trans. Roy. Soc. Trop. Med. & Hyg., 31:549 (1938).

102. Jones, M. F. and Hummel, M. S. Studies on Schistosomiasis: The Effect of Chlorine and Chloramine on Schistosome Ova and Miracidia. Nat. Inst. Health Bull. No. 189, Washington (1947).

References (continued)

103. Barlow, C. H. The Value of Canal Clearance in the Control of Schistosomiasis in Egypt. Am. J. Hyg., 25:327 (1937).

104. Barlow, C. H. Further Studies of the Revival, After Drying, of the Snail Hosts of the Human Schistosomes of Egypt. Am. J. Hyg., 22:376 (1935).

105. Chernin, E., Michelson, E. H. and Augustine, D. L. Studies on the Biological Control of Schistosome-bearing Snails. I. The Control of *Australorbis glabratus* Populations by the Snail, *Marisa cornuarietis,* under Laboratory Conditions. Am. J. Trop. Med. & Hyg., 5:297 (1956).

106. Ferguson, F. F. and Palmer, J. R. Biological Notes on *Marisa cornuarietis,* A Predator of *Australorbis glabratus,* the Snail Host of Schistosomiasis in Puerto Rico. Am. J. Trop. Med. & Hyg., 7:640 (1958).

107. Study Group on the Ecology of Intermediate Snail Hosts of Bilharziasis. Technical Report Series, No. 120. World Health Organization, Geneva (1957).

108. Expert Committee on Bilharziasis. Technical Report Series, No. 65. World Health Organization, Geneva (1953).

109. Hunter, G. W., Frye, W. W. and Swartzwelder, J. C. A Manual of Tropical Medicine. W. B. Saunders Co., Philadelphia (3rd ed., 1960).

110. Klatskin, G., Amebiasis of the Liver: Classification, Diagnosis and Treatment. Ann. Int. Med., 25:601 (1946).

111. Hegner, R., Beltran, E. and Hewitt, R. Human Intestinal Protozoa in Mexico. Am. J. Hyg., 32:27 (1940).

112. Sapero, J. J. and Johnson, C. M. Endameba Histolytica and Other Intestinal Parasites: Incidence in Variously Exposed Groups of the Navy. U. S. Navy. Med. Bull., 37:279 (1939).

113. Wenrich, D. H., Stabler, R. M. and Arnett, J. H. Endamoeba Histolytica and Other Intestinal Protozoa in 1,060 College Freshmen. Am. J. Trop. Med., 15:331 (1935).

114. Burrows, R. B., Prevalence of Amebiasis in the United States and Canada. Am. J. Trop. Med. & Hyg., 10:172 (1961).

115. Ivanhoe, G. L. Studies on the Transmission of Amebiasis in a Children's Home in New Orleans. Am. J. Trop. Med., 23:401 (1943).

116. Sapero, J. J. and Johnson, C. M. An Evaluation of the Role of the Food Handler in the Transmission of Amebiasis. Am. J. Trop. Med., 19:255 (1939).

117. Roberts, E. W. The Part Played by the Faeces and Vomit-drop in the Transmission of Entamoeba Histolytica by Musca Domestica. Ann. Trop. Med. & Parasit., 41:129 (1947).

118. Pipkin, A. C. Filth Flies as Transmitters of Endamoeba Histolytica. Proc. Soc. Exper. Biol. & Med., 49:46 (1942).

119. Manson-Bahr, P. The Dysenteric Disorders. Cassell and Co., Ltd., London (2nd ed., 1944).

120. Bundesen, H. N., Connolly, J. I., Rawlings, I. D., Gorman, A. E., McCoy, G. W. and Hardy, A. V. Epidemic Amebic Dysentery. Nat. Inst. Health Bull. No. 166 Washington (1936).

121. Fassnacht, G. G. and Fooks, J. H. Engineering Studies on Amebiasis Outbreak at South Bend. Jour. AWWA, 46:1129 (1954).

122. Spector, B. K., Baylis, J. R. and Gullans, O. Effectiveness of Filtration in Removing from Water and of Chlorine in Killing, the Causative Organism of Amoebic Dysentery. Pub. Health Rep., 49:786 (1934).

123. Baylis, J. R., Gullans, O. and Spector, B. K. The Efficiency of Rapid Sand Filters in Removing the Cysts of the Amoebic Dysentery Organisms from Water. Pub. Health Rep., 51:1567 (1936).

124. Fair, G. M., Morris, J. C. and Chang, S. L. The Dynamics of Water Chlorination. Jour. N. England Wat. Wks. Assoc., 61:285 (1947).

125. Chang, S. L. and Morris, J. C. Elemental Iodine as a Disinfectant for Drinking Water. Indust. & Eng. Chem., 45:1009 (1953).

References (continued)

126. Eichenwald, H. F. Viral Hepatitis. U. S. Public Health Service Publication No. 435 (1955).

127. Cameron, J. D. S. Infective Hepatitis. Quart. J. Med., 12:139 (1943).

128. MacCallum, F. O. and Bradley, W. H. Transmission of Infective Hepatitis to Human Volunteers. Lancet, II:228 (1944).

129. Roos, Bertil. Hepatitis Epidemic Conveyed by Oysters. Svenska Lakartidningen, 53:989 (1956).

130. Rindge, M. E. Infectious Hepatitis and Its Relation to Polluted Shellfish. Connecticut Health Bull., 75:217 (1961).

131. Murphy, W. J., Petrie, L. M. and Work, S. D. Outbreak of Infectious Hepatitis, Apparently Milk-borne. Am. J. Pub. Health, 36:169 (1946).

132. Read, M. R., Bancroft, H., Doull, J. A. and Parker, R. F. Infectious Hepatitis—Presumably Food-borne Outbreak. Am. J. Pub. Health, 36:367 (1946).

133. Knight, V., Drake, M. E., Belden, E. A., Franklin, B. J., Romer, M. and Copple, L. O. Characteristics of Spread of Infectious Hepatitis in Schools and Households in an Epidemic in a Rural Area. Am. J. Hyg., 59:1 (1954).

134. Davis, D. J. and Hanlon, R. C. Epidemic Infectious Hepatitis in a Small Iowa Community. Am. J. Hyg., 43:314 (1946).

135. Mosley, J. W. and Smither, W. W. Infectious Hepatitis. N. England J. Med., 257:590 (1957).

136. Fraser, R. A Study of Epidemic Catarrhal Jaundice. Canad. J. Pub. Health, 22:396 (1931).

137. Neefe, J. R. and Stokes, J., Jr. An Epidemic of Infectious Hepatitis Apparently Due to a Water Borne Agent. J.A.M.A., 128:1063 (1945).

138. Hayward, M. L. Epidemiological Study of an Outbreak of Infectious Hepatitis. Gastroenterology, 6:504 (1946).

139. Viswanathan, R. Infectious Hepatitis in Delhi (1955-1956): A Critical Study. Ind. J. Med. Res., 45, Supp. Number: 1 (1957).

140. Neefe, J. R., Baty, J. B., Reinhold, J. G. and Stokes, J., Jr. Inactivation of the Virus of Infectous Hepatitis in Drinking Water. Am. J. Pub. Health, 37:365 (1947).

141. Messih, G. A. Head, Department of Epidemiology, The High Institute of Public Health, United Arab Republic, Alexandria, Egypt. Mimeographed paper (1960).

142. Hobbs, B. C., Smith, M. E., Oakley, C. L., Warrack, G. H. and Cruickshank, J. C. *Clostridium Welchii* Food Poisoning. J. Hyg., 51:75 (1953).

143. Zobel, D. C. Analysis of Data on Revised Death Certificates in Venezuela. Mimeographed paper. Oficina Cooperativa Interamericana de Salud Publica, Caracas (1957).

144. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, 7th Revision, 1955. World Health Organization, Geneva (1957).

145. Verhoestraete, L. J. and Puffer, R. R. Diarrhoeal Disease with Special Reference to the Americas. Bull., 19:23, World Health Organization, Geneva (1958).

146. Huebner, R. J., Risser, J. A., Bell, J. A., Beeman, E. A., Beigelman, P. M. and Strong, J. C. Epidemic Pleurodynia in Texas. N. England J. Med., 248:267 (1953).

147. Melnick, J. L., Emmons, J., Coffey, J. H. and Schoof, H. Seasonal Distribution of Coxsackie Viruses in Urban Sewage and Flies. Am. J. Hyg., 59:164 (1954).

148. Kelly, S. Enteric Virus Isolations from Sewage. Acta med. scandinav., 159:63 (1957).

149. Kelly, S. M., Clark, M. E. and Coleman, M. B. Demonstration of Infectious Agents in Sewage. Am. J. Pub. Health, 45:1438 (1955).

References (continued)

150. Clarke, N. A., Stevenson, R. E. and Kabler, P. W. Survival of Coxsackie Virus in Water and Sewage. Jour. AWWA, 48:677 (1956).

151. Chang, S. L., Stevenson, R. E., Bryant, A. R., Woodward, R. L. and Kabler, P. W. Removal of Coxsackie and Bacterial Viruses in Water by Flocculation. II. Removal of Coxsackie and Bacterial Viruses and the Native Bacteria in Raw Ohio River Water by Flocculation with Aluminum Sulfate and Ferric Chloride. Am. J. Pub. Health, 48:159 (1958).

152. Clark, N. A. and Kabler, P. W. The Inactivation of Purified Coxsackie Virus in Water by Chlorine. Am. J. Hyg., 59:119 (1954).

153. Rivers, T. M. and Horsfall, F. L. Viral and Rickettsial Infections of Man. J. B. Lippincott Co., Philadelphia (3rd ed., 1959).

154. Aycock, W. L. A Milk-borne Epidemic of Poliomyelitis. Am. J. Hyg., 7:791 (1927).

155. Knapp, A. C., Godfrey, E. S. and Aycock, W L. An Outbreak of Poliomyelitis—Apparently Milk Borne. J.A.M.A., 87:635 (1926).

156. Trask, J. D. and Paul, J. R. The Detection of Poliomyelitis Virus in Flies Collected During Epidemics of Poliomyelitis. J. Exper. Med., 77:545 (1943).

157. Melnick, J. L. and Penner, L. R. The Survival of Poliomyelitis and Coxsackie Viruses Following Their Ingestion by Flies. J. Exper. Med., 96:255 (1952).

158. Paul, J. R. and Trask, J. D. The Virus of Poliomyelitis in Stools and Sewage. J.A.M.A., 116:493 (1941).

159. Rhodes, A. J., Clark, E. M., Knowles, D. S., Shimada, F., Goodfellow, A. M., Ritchie, R. C. and Donohue, W. L. Poliomyelitis Virus in Urban Sewage. Canad. J. Pub. Health, 41:248 (1950).

160. Chang, S. L. Viruses, Amebas, and Nematodes and Public Water Supplies. Jour. AWWA, 53:288 (1961).

161. Krumbiegel, E. R. Transmission of Virus Disease by Water. Jour. AWWA, 36:81 (1944).

162. Maxcy, K. F. Supposed Involvement of Water Supplies in Poliomyelitis Transmission. Jour. AWWA, 41:696 (1946).

163. Little, G. M. Poliomyelitis and Water Supply. Canad. J. Pub. Health, 45:100 (1954).

164. Bancroft, P. M., Englehard, W. E. and Evans, C. A. Poliomyelitis in Huskerville (Lincoln), Nebraska. J.A.M.A., 164:836 (1957).

Relationship of WATER to OTHER DISEASES

Other infectious agents are a threat to man's well being. Some of these require water to maintain their life cycle and to serve as their habitat; others in the egg form are available, sometimes in large numbers, for transmission by water. Man must recognize the menace of these agents and the mode of their dissemination if he is to combat their threat.

WATER-BRED PLANTS AND ANIMALS. Certain insects, worms, plants, fish and crustacea need water to continue their life cycle and to live in while becoming dangerous to man. The condition of the water—quiet, running, natural, artificial, clean, polluted, fresh—bears upon its selection for inhabitation by the plant or animal life involved, in some way, in disease transmission.

The mosquito, with which the world is very familiar, could not exist without water. As it is, this insect viciously implants viruses in man to cause such diseases as dengue, encephalitis, Rift Valley fever and yellow fever; it carries to him both the protozoa of malaria and the nematode responsible for filariasis.

The larvae of certain flies are aquatic and need water for their continued existence. For example, flies of the genus *Chrysops* and of the genus

Simulium (black fly) are in this group. *Chrysops* leaves in man, after the bite, infected larvae of a nematode which causes loaiasis and *Simulium* performs the same action with another nematode which infects man with onchocerciasis.

Using fresh-water fish and crustacea (crabs and crayfish) as the immediate sources of infection, trematodes and a cestode perpetuate certain diseases. Clonorchiasis is caused by a fluke and diphyllobothriasis by a tapeworm, and both reach man by way of the ingestion of parasitized fish. In a similar manner, crabs and crayfish infected with a trematode produce paragonimiasis in man. More directly, man may consume fluke cercariae responsible for fasciolopiasis when he eats the aquatic plant, water chestnut.

ROUTES OF INFECTIVE AGENTS. The infective agents of the following diseases are not carried to man by water but water plays an important part in the propagation of the vehicles of their transmission. The avenues usually traversed by the infecting organisms are illustrated below:

Clonorchiasis:	animals / man } snail—fish—man
Dengue:	man—mosquito—man
Diphyllobothriasis:	animals / man } crustacean—fish—man
Encephalitis:	animals—mosquito—man
Fasciolopiasis:	animals / man } snail—aquatic plant—man
Filariasis:	man—mosquito—man
Loaiasis:	man—fly—man
Malaria:	man—mosquito—man
Onchocerciasis:	man—fly—man
Paragonimiasis:	animals / man } snail— crab / crayfish } man
Rift Valley fever:	animals—mosquito—man
Yellow fever:	animals / man } mosquito—man

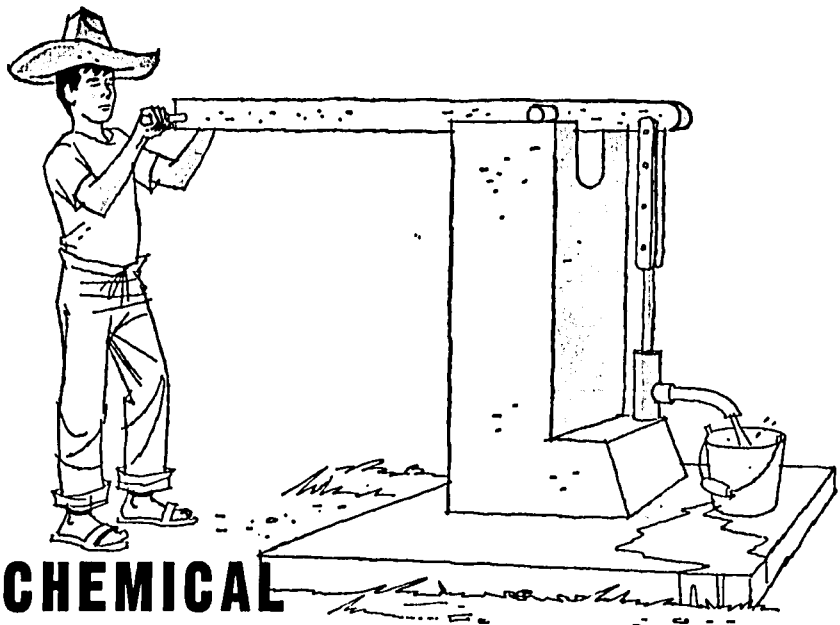
OTHER HELMINTHIC DISEASES. Four intestinal infections—ascariasis, trichuriasis, enterobiasis and ancylostomiasis—are caused by nematodes.

The eggs of the roundworms *Ascaris lumbricoides* (ascariasis) and *Trichuris trichiura* (trichuriasis) leave the infected person in the feces. Indiscriminate defecation by infected individuals results in the deposition of

the eggs on the soil where embryonation takes place. If these eggs are ingested, either directly from the soil or by means of vehicles including water, infection may result.

The human whipworm, *Enterobius vermicularis* (enterobiasis), discharges its eggs on the perianal skin. This is done by the gravid worms following their migration to the rectum. The sources of infection with these eggs are generally clothing, bedding, food and other contaminated articles but dust-borne eggs may find their way into water which is subsequently consumed. Ingestion of the eggs is necessary to produce the infection.

Necator americanus and *Ancylostoma duodenale* are the two round-worms (hookworms) responsible for most of the cases of ancylostomiasis. When the feces of an infected person are deposited on a favorable soil and under advantageous conditions, the eggs will hatch and larvae will emerge and develop into the filariform stage which is infective to man. While water may serve as a medium to bring about the swallowing of mature filariform larvae which will then develop into worms, infection is usually acquired by skin penetration.



CHEMICAL SUBSTANCES in WATER

Even the purest natural waters are far from pure either from the chemical* or the physiological standpoint but contain suspended matter and dissolved substances in varying amounts. Among the common impurities in water are plant and animal life and soil particles in suspension, and minerals, gases, and plant extracts in solution.

Domestic use of aesthetically satisfactory water that has not previously been employed by man for agricultural or industrial purposes, or for receiving wastes is more likely to be hazardous to health because of the presence of micro-organisms** than of chemicals. Following indiscriminate or multipurpose use, however, pollution by a variety of chemicals, as well as by living organisms, may increase the health hazard.

Studies made on ground and surface waters following usage have revealed a multiplicity of chemical substances not present before such usage. Some have been toxic as illustrated by the findings of research on the Ohio

* The chemical elements, compounds or materials included in this chapter parallel those deemed to merit consideration in the U.S. Public Health Service Drinking Water Standards.[165]

** Discussed in preceding chapters.

River (U.S.) where phenols, and chromium and copper salts were detected.[166] More recent data on fishkills along 600 miles of rivers and 5,000 acres of lakes in the United States, obtained for the summer of 1960, showed 185 such incidents of which 73 were attributed to agricultural poisons, 57 to industrial wastes and the remainder to other causes.[167]

Toxic substances may be found in water due to the presence of water-blooms. Davidson [168] has reported that wild and domestic animals became acutely ill after drinking water containing a heavy growth of *Nostoc rivulare* Kuetz and that many died within a short period of time. Under experimental conditions, crude and unpurified algae material has been lethal to mice, the nature of the toxin not being determined.[169] Several thousand cases of intestinal disorders occurred in Charleston, West Virginia (U.S.) in 1931 and a heavy algae growth present in a very polluted pool, the source of water supply remaining in an exceptionally dry period, was suspected of contributing to this outbreak although this was never proven.[170]

Some of the chemicals in water may be merely aesthetically unpleasant or distasteful, serving mainly as deterrents to its free and full use. Other chemicals may be capable of reacting to form substances with altered toxicity and still others may be beneficial to animal and human health.

The chemical substances treated in this chapter are divided into two groups. Group I includes those known to possess toxic qualities when present in drinking water at levels above specific limits and others which appear to have a similar potential based upon evidence of their toxicity when conveyed to man in media other than water. None of these substances should be permitted to exceed the limits set forth if there is to be full assurance that health effects will not occur among the consumers of water containing these chemicals.

Group I

1. Arsenic
2. Barium
3. Cadmium
4. Chromium, hexavalent
5. Cyanide
6. Fluoride
7. Lead
8. Selenium
9. Silver

ARSENIC (As). Traces of arsenic are fairly common in natural waters and, as an arsenate, it has been found in hot springs.[171] Contributions of arsenic to surface waters may be made by industrial and mining activities or by the surface-wash of lands where insecticides and herbicides have been employed. However, Neal [172] has reported no significant contamination of water supplies coming from the watersheds where lead arsenate was used as a pesticide spray by agriculturists. The tolerance for arsenic on sprayed fruits and vegetables in the United States is 3.5 ppm of combined As_2O_3 in terms of the weight of the specimen examined.[173]

Arsenic compounds are very toxic and as little as 100 mg usually causes severe poisoning when ingested. Chronic arsenic poisoning is deceptive and pernicious; the slow excretion of arsenic is in part the reason for its cumulative toxic effects. Deaths among cattle in New Zealand have been reported as a result of drinking natural water containing arsenic.

There is some evidence pointing to the belief that arsenic may be carcinogenic. Employees of an arsenical powder manufacturing plant who were exposed to arsenic dust showed a higher incidence of skin and lung cancer than other occupational groups.[174 175]

In view of the present knowledge concerning the potential health hazard from ingesting inorganic arsenic, the U.S. Public Health Service Drinking Water Standards [105] recommends the rejection of drinking water containing more than 0.05 ppm,* and the avoidance of the use of a water supply containing a concentration of more than 0.01 ppm if another more suitable supply is available at a reasonable cost.

BARIUM (Ba). In ground and stream waters, only small amounts of this element are present. Piper [176] reports finding barium in connate waters largely associated with wells in oil fields. Braidech [177] made spectrographic determinations of minor chemical constituents in the water supplies of 24 cities in the United States and recorded that the barium content varied from 0.04 ppm to 0.5 ppm.

Barium is known to be a general muscle stimulant. Evidence exists for high acute toxicity of ingested soluble barium salts and for chronic irreversible tissue changes from the deposit of insoluble forms of barium in sufficient amount at a localized site. However, no accumulation of barium has been reported to occur in bone, muscle, or kidney from experimentally administered barium salts in animals.

Information regarding the amount of barium that may be tolerated in drinking water is not available. Neither are there data on the effects from prolonged feeding of barium salts to support an acceptable water standard. Utilizing the threshold limit for air of 0.5 mg per cubic meter,[178] Stokinger and Woodward [179] reached the conclusion that 1.0 ppm in water constituted the limit of the "no effect" level but in their rationalization, they made reasonable assumptions regarding the retention of inhaled barium dusts and absorption from the intestine, and provided a safety factor.

Because of the seriousness of the toxic effects of barium on the heart, blood vessels and nerves, the rejection of drinking water containing barium in excess of 1.0 ppm is advised.[105]

CADMIUM (Cd). Cadmium is likely to be present in natural waters only in minute traces but it may be introduced in amounts significant to health by solution from containers or tubing, or by waste products. In the dissolved state, cadmium is a powerful emetic and a gastrointestinal irritant, and in the form of its soluble salts it can cause both acute and chronic poisoning.

Frant [180] reported on four outbreaks of cadmium poisoning which resulted from the contamination of certain foods and drink by soluble cadmium compounds. The food item in one of the incidents contained 530 ppm of cadmium and cadmium-plated utensils were involved in all the outbreaks. Coffee and lemonade held in cadmium-plated containers for periods of 1½ to 14 hours produced several large cadmium poisoning outbreaks among military personnel, according to Cangelosi.[181]

* A ppm is equivalent to a milligram per liter.

Cadmium has been described as a water contaminant by Lieber[182] who studied the penetration of the soil by cadmium-containing wastes stored for disposal in ground-recharging basins on Long Island, New York. In two shallow wells, he found 0.6 ppm and 0.34 ppm respectively and test wells revealed concentrations up to 3.2 ppm, the amount depending upon the location of the well with respect to that of the waste disposal site and the depth at which the sample was taken.

In a study designed to determine the effects of drinking water contaminated with cadmium, Decker and his associates[183] gave five groups of rats drinking water containing it at levels from 0.1 to 10 ppm. No toxic effects were seen but the cadmium content of the liver or the kidney increased in direct proportion to the cadmium intake. Tissue concentrations at the end of a year were double those at the end of six months. Toxic effects were seen in rats ingesting 50 ppm for three months. In other experiments, Ginn[184] learned that 50 ppm of cadmium as cadmium chloride, given to rats in food and drinking water, reduced blood hemoglobin and lessened dental pigmentation.

All levels of dietary cadmium so far tested have shown cadmium accumulation in the soft tissues and the presence of minute amounts of cadmium in rat liver mitochondria[185] has been shown to interfere with an important pathway of metabolism. Also there is suggestive but unproven evidence that cadmium is related to chronic sustained arterial hypertension.[186]

In light of existing evidence against cadmium, water containing it in excess of 0.01 ppm should be rejected as unsuitable for domestic consumption.[165]

CHROMIUM (Hexavalent Cr). Very little chromium in igneous rocks goes into solution; hence, natural waters would be expected to contain only traces of it as a cation, unless the pH were very low. Braidech[177] cited the chromium content of certain public water supplies in the United States as varying from 0.001 ppm to 0.04 ppm.

Surface waters are subject to contamination with chromium compounds coming from industrial processes. Graham[187] found that the Los Angeles River (U.S.) received wastes of this type from manufacturing plants performing chromium plating.

Davids[188] reported that hexavalent chromium was to be found in ground waters of Long Island, New York (U.S.) in 1942, presumably coming from storage basins holding industrial wastes. One private well examined which had been used by a family of four for at least three years prior to the time of sampling with no detectable ill effects was found to contain 1.0 ppm. Three years later (1951) this well contained 25 ppm and the family was still using it. Test wells drilled in an effort to define the area of underground contamination showed up to 40 ppm of chromium.

An analysis of mortality data of exposed employees in the United States chromate-producing industry by Machle[189] revealed a high death rate from cancer of the respiratory system. Whether hexavalent chromium—or chromium of other valence—is cancerigenic to man if taken by the oral route is not known. Trivalent chromium in drinking water is believed to be of no hygienic significance.[190]

MacKenzie and his associates [191] fed water containing between 0.45 ppm and 11 ppm of hexavalent chromium to five groups of rats for a year. There were neither differences between these rats and the control groups as to water intake, food consumption or weight gain nor were there any changes found upon blood analysis. There was a rise in tissue chromium concentration when the rats drank water containing amounts of chromium ion in excess of 5.0 ppm. In another test, water containing 25 ppm of hexavalent and trivalent chromium fed to two groups of rats respectively for one year produced no toxic symptoms. Growth curves for white rats calculated following experimental work by Gross [192] indicated that the maximum non-toxic level of chromate salts in drinking water is 500 ppm.

In light of the evidence presently available, a concentration of 0.05 ppm in drinking water is believed to be sufficiently low to result in no toxic effects if consumed at that or a lower level for a lifetime.[165]

CYANIDE (CN). Cyanides are common to many modern industrial processes and they are soluble in water. Hence, they may be found in raw waters receiving industrial wastes from specific industrial processes.

Smith [193] reports that 2.9-4.7 mg of cyanide per day, taken orally, is not injurious and Bodansky [194] says that neither is 10 mg in a single dose. Cyanide, in reasonable doses (10 mg or less), is detoxified in man's liver and when toxic effects are lethal it is usually because the detoxifying mechanism is overwhelmed. Fish are quite sensitive to cyanide in water and can serve as an indicator of its presence. Karsten [195] states that concentrations over 1.0 ppm are certain to kill all trout within 20 minutes.

For the protection of the health of human populations, drinking water containing more than 0.2 ppm cyanide should not be accepted or used.[165] Proper chlorination under neutral or alkaline conditions will reduce cyanide by the formation of cyanogen chloride, a substance with an acute toxicity of one-twentieth of that of hydrogen cyanide, when taken by mouth. A limit of 0.01 ppm is appropriate when a more suitable source of water potentially free of cyanide, can be made available at a reasonable cost.[165]

FLUORIDE (F). Fluoride concentration in ground waters may range up to 50 ppm or more but more than 10 ppm is rare and surface waters seldom contain more than 1.0 ppm.[171] Sampling and subsequent testing of surface and well waters in a large number of communities of the United States by Kehoe [196] revealed a mean concentration of 0.317 ppm.

Excessive fluoride in drinking water causes dental fluorosis (mottled enamel) which increases in the intensity of its effect with increasing fluoride concentrations. Following observation of this condition, Dean [197] suggested that the mineral composition of drinking water might have an important bearing on the incidence of dental caries. His examination of nine-year old children with continuous exposure showed a higher percentage of children to be caries-free in those communities where the domestic water supplies contained a higher concentration of fluorides in comparison with communities using water having a lower concentration.

Hodge has reported [198] that 13 of 114 residents of Bartlett, Texas (U.S.) where the drinking water contained 8.0 ppm of fluoride showed osteosclerosis,

and that 21 of 178 of those living in a specific South American area and who were drinking water having in it 16 ppm, gave evidence of bone changes by x-ray examination. A single-dose intake of fluoride in the range of 2,250-4,500 mg can be lethal.

Continued study of the relationship of fluoride in water to dental fluorosis and caries, finally resulted in the concept that controlled concentrations of fluorides in drinking water might be used to the advantage of man. Dean and his associates,[199] following an extended study of 7,257 white urban children aged 12 to 14 years continually exposed to the drinking water supply of their respective home community, found that there was a general inverse correlation between the fluoride concentrations of the public water supplies and the amount of dental caries observed and that low dental caries prevalence, without the harmful effects of fluorosis, was associated with the continuous use of domestic waters having a fluoride content of about 1.0 ppm.

With the initiation and extension of the practice of supplementing fluoride intake to the optimum level, using drinking water as the vehicle, Galagan [200] pointed out that water intake—and hence the fluoride intake—increased directly with increases in air temperature under normal living conditions in the United States.

The effectiveness of fluoride in preventing dental caries was confirmed by the 10-year observation of participants in a study in Michigan.[201] Here the adjustment of the fluoride content of the communal water supply to 1.0 ppm produced a reduction of the caries rate for the deciduous teeth in 6-year olds by 54 per cent and, in children born subsequent to the start of the fluoridation program, the caries rate in permanent teeth was reduced by 60 per cent.

There are abundant data showing the desirability of altering drinking water so that it will contain an amount of fluoride suitable for the best tooth development. Changing the source of drinking water supply, supplementing the fluoride content of an existing one by the mechanical addition of fluoride compounds, and partial defluoridation to remove fluoride present in excess of an optimum amount [202] are all means to improve man's physical condition.

The U.S. Public Health Service Drinking Water Standards specify that when fluoride is naturally present in drinking water, the concentration should not average more than the appropriate upper limit in table 9 and drinking

TABLE 9
Fluoride Concentrations [165]

Annual Average of Maximum Daily Air Temperatures in Degrees F.[a]	Limits of Fluoride Concentrations in ppm		
	Lower	Optimum	Upper
50.0—53.7	0.9	1.2	1.7
53.8—58.3	0.8	1.1	1.5
58.4—63.8	0.8	1.0	1.3
63.9—70.6	0.7	0.9	1.2
70.7—79.2	0.7	0.8	1.0
79.3—90.5	0.6	0.7	0.8

[a] Based on temperature data obtained for a minimum of five years.

water should be rejected as inimical to man's health if average concentrations are greater than twice the optimum values in that table. When fluoride in drinking water is artificially supplemented by the controlled addition of fluoride compounds (fluoridation), the average concentration should be kept within the upper and lower limits in table 9.

LEAD (Pb). Only traces of lead are likely to be found in natural waters. Kehoe [196] reports data showing the presence of lead in natural surface and well waters in quantities ranging from 0.001 ppm to 0.06 ppm; his own tests on the drinking water of a number of localities in the United States showed lead in amounts from 0.001 to 0.04 ppm.

There is ample evidence, however, that in the past the lead content of drinking water has been measurably increased by the solution of lead from lead pipes through the action of aggressive water. For this reason, the use of such pipe to conduct water likely to corrode it should be avoided. In the period of 1909-1910, 49.5 per cent of 699 examinations of drinking water for lead made by Howard [203] showed its presence in 0.5 ppm or more; in 1921-1922, 18.7 per cent of 380 tests gave the same results. Howard states that most of the samples were of ground waters and lead pipe was almost invariably used to conduct the water. Wright [204] studied clinically 253 persons using water from 90 different lead-conducted water supplies. All the waters tested contained lead and 35 of the supplies were responsible for lead poisoning. The presence of a lead line or the stippling of red blood cells, and at least two other symptoms common to lead poisoning were indicative of mild lead poisoning in 24.9 per cent of those studied.

Negus [205] has cited cases of plumbism (lead poisoning) which were traced to the consumption of water from streams and wells located near regions in which heavy agricultural spraying with lead arsenate was carried on. Neal [172] found that irrigation water passing through orchard areas where lead arsenate was used as a spray contained only 0.003 to 0.071 ppm of lead.

Other main sources from which man may unintentionally acquire lead are food, air and tobacco smoke. According to Kehoe,[206] a resident of the United States accretes an average of 0.32 mg of lead per day from food and beverage. At this level, the body's excreting process is adequate to preclude a significant accumulation but if the daily intake rises to more than 0.6 mg, the rate of the body's accumulation will increase as the daily dose increases.

Other studies by Kehoe [207] indicated that a resident of a specific large city in the United States will breathe in about 0.1 mg of lead each day. The extent to which this is absorbed is unknown but it is conjectured to amount to 10 per cent.

The lead contained in the foods used by man is natural or incidental, and its presence is largely unavoidable. Lead is in much of the atmosphere which man must breathe and it has increased in quantity more than 10-fold over the last decade in urban communities in the United States. Heavy cigarette smokers add to the lead body burden with each cigarette puff. Indicated but unproven is the fact that a lead intake over a five-year period appreciably in excess of 0.6 mg per day will result in the accumulation of

a dangerous quantity of lead in the body during a lifetime. Hence, lead in drinking water should be held below 0.05 ppm and water containing more than that should be considered unacceptable for human consumption.[165]

SELENIUM (Se). Natural surface waters have been found to contain up to 2.5 ppm and natural ground waters up to 1,600 ppm of this element by Kehoe and his coworkers.[196]

In some regions, the presence of selenium in soils and plants has presented a problem in livestock management. Cattle feeding on plants containing up to 25 ppm of selenium suffer the chronic "alkali disease" and plants containing 100 to 1,000 ppm may produce in them the acute disease "blind staggers." [208] In animals (hogs, calves, sheep, horses) fed inorganic or organic selenium compounds, selenium is found generally in the liver, kidneys and spleen in the acute stage of the poisonings and in the chronic stage it is predominantly in the liver and kidneys.[209]

Fitzhugh [210] reports that rats fed selenium in a grain diet at six concentrations varying from 3 to 40 ppm showed toxic effects at all selenium levels. The main pathological lesion found was cirrhosis of the liver; among 43 cirrhotic rats which survived 18 months or longer, hepatic cell tumors developed in 11.

Mild chronic selenium poisoning has been seen in man living in seleniferous regions and toxic symptoms have been observed in persons breathing hydrogen selenide at a level as low as 0.2 ppm.[208] Hadjimarkos [211] found a correlation between urinary selenium and the prevalence of dental caries in school children living in seleniferous localities and he concluded that this element may be a factor bearing upon the increase of susceptibility to caries attack.

Because of the potential seriousness of the result of selenium on man, particularly the possible carcinogenic effect, selenium in drinking water should not be permitted to rise to a quantity in excess of 0.01 ppm.[165]

SILVER (Ag). Silver is not likely to be found in any large amount in natural waters. One investigator's [196] examinations showed only traces of it in samples of natural surface and ground waters.

The need to protect the health of water consumers against adverse physiological effects of silver derives from its intentional addition as a disinfecting agent. Silver added to water acts in a certain definite way on bacteria, and on the microfauna and microflora in the water. Part of the added silver remains ionized for a considerable period of time and conveys thereby a germicidal property to it which serves a useful purpose in combatting any secondary contamination, according to Just.[212] However, following his experimental work with silver-fed rats in 1936, he concluded that the use of silver in water for disinfecting purposes could not be considered a procedure innocuous to health. Just and his coworker fed eight groups of rats drinking water containing doses of silver ranging from 0 to 1,000 ppb.* All rats received the same food and were held under the same conditions. Histological examination showed changes in the kidneys, liver and spleen in

* ppb=part per billion.

those which had received 400, 700 and 1,000 ppb. These changes became more distinct as the dose grew larger.

The deposition of a silver compound in the body results in argyria which develops gradually when the silver enters the tissues by direct injection, continued application to mucous surfaces, or long-time oral ingestion. It is practically incurable.[213] The amount of colloidal silver needed to produce this condition is unknown. However, Hill[214] learned from studying cases of argyria that the smallest amount of metallic silver in silver arsphenamine, administered intravenously, which did cause this condition was 0.91 g. Hill[214] states that there is "abundant evidence that silver is deposited in the majority of the structures of the body in argyria with the possible exception of the nerve tissues and muscles. The involved organs show no pathologic alteration that can be attributed to silver." Once silver is fixed in the tissues, little excretion of it in the urine takes place.

When silver-treated water is used for cooking certain vegetables, it is not unreasonable to think that these vegetables would combine with the silver in the water and thus convey silver to the consumer. However, great uncertainty is attached to an evaluation of the amount of silver introduced into the body in this way. Kehoe[215] reports finding 0.06-0.08 mg of silver per day in feces and food.

If it is assumed that all the silver contained in ingested drinking water is deposited in the skin and that approximately two liters of water is consumed each day by a person, it can then be calculated that one could drink 0.01 ppm per day for a lifetime and 0.05 ppm for about 27 years without exceeding a deposition in the skin of 1.0 g. Therefore, the silver content of drinking water should not exceed 0.05 ppm.[165]

The members of the second group of substances are of varying physiological importance but have definite bearing upon the acceptability of the water for domestic use. Water containing these substances in quantities exceeding the limits subsequently shown will be objectionable to many people. If water containing any of these constituents can be avoided by a reasonable and economical shift to another source of raw water supply, by appropriate treatment or by other quality control practices, such change is recommended.

Group II

1. Alkyl Benzene Sulfonate
2. Carbon Chloroform Extract
3. Chloride
4. Copper
5. Iron
6. Manganese
7. Nitrate
8. Phenols
9. Sulfate
10. Total Dissolved Solids
11. Zinc

ALKYL BENZENE SULFONATE (ABS). Synthetic detergents or surface-active agents are cleansing compounds of organic chemical structure which have come into widespread use. Chemically, there are two types of them, ionic and nonionic, and the ionic type may be either anionic or cationic. In the United States, probably 75 per cent of the surfactants in household detergents are of the anionic type and alkyl aryl sulfonates will comprise three-fourths of these, the remainder being mostly alkyl sulfates.

Alkyl benzene sulfonate is the sodium salt of commercial sulfonated dodecylbenzene; it is the most commonly used synthetic surface-active agent in household detergents and it is material of this variety which is most likely to be found in raw waters receiving wastes from homes and other places.

Walton [216] has reported that samples of Illinois River (U.S.) water taken at ten different locations showed apparent ABS concentrations from 0.5 ppm to 1.3 ppm. He has also summarized the results of many studies of ABS concentrations in water supplies in housing developments where each unit had its own well and subsurface sewage disposal system. In the 976 well waters examined, 357 had ABS concentrations of at least 0.1 ppm and, in three cases, it was 4.0 ppm.

Man may also consume detergents adhering to inadequately cleansed household utensils and dishes.

Drinking water containing as little as 2.0 ppm of surfactants can cause a discernible taste which chlorine may intensify and then remove [217] and frothing has been reported to have occurred at a concentration as low as 0.6 ppm.[218]

There appears to be little specific relationship of toxicity to surfactant activity.[219] Fitzhugh and Nelson [220] studied the chronic oral toxicity of several surface-active agents including sodium alkyl aryl sulfonate and concluded that the toxic effects were produced by irritation of the gastrointestinal tract; irritation prevented proper nutrition. A 2 per cent concentration of this agent in the diet of rats caused a significant decrease in their rate of growth and 1 per cent (10,000 ppm) slightly retarded it. Freeman [221] learned that the ingestion by humans of 100 mg of purified sodium alkyl aryl sulfonate daily for four months led to no significant evidence of intolerance.

Concentrations of ABS above 0.5 ppm are indicative of an undesirable level of sewage pollution and this fact, coupled with the tendency of drinking water to foam when containing 1.0 ppm, points to the desirability of establishing 0.5 ppm as the limit above which ABS should not be permitted to rise in domestic water.[165]

CARBON CHLOROFORM EXTRACT (CCE). Procedures are available for the determination of the inorganic compounds in water. The identification and measurement of specific organic compounds are more demanding tasks and it is even doubtful if a complete analysis of them could presently be made.

Synthetic organic substances have grown in number at a very rapid rate since about 1940 until today thousands are being used in our living and manufacturing processes. Many, of course, find their way into waterways and are now present in several major streams in the United States in concentrations up to 0.5 ppm.[222] Recent studies [223] have shown the CCE content of samples of finished drinking waters coming from 64 surface water sources in the United States to be as follows: 27 ranging from 0 to 0.05 ppm; 28 from 0.05 to 0.1 ppm; 7 from 0.1 to 0.15 ppm; and 2 from 0.15 to 0.2 ppm.

Records do not reveal any human illness attributable to these complex compounds in drinking water but past experience is not a guarantee that this

situation will prevail in the future because it must be assumed that human tolerance to these materials is not unlimited.

Total absence of organic residues from drinking water is the most desirable condition; when they are present, it means that man-made or natural pollutants are not being removed from the raw water by the treatment processes in use or that some secondary contamination is taking place. In view of the present inability to clearly characterize and interpret the chemical and toxicological nature of organic residues, it is desirable to limit them to the lowest level attainable and not to exceed 0.2 ppm.[165]

CHLORIDE (Cl). See Total Dissolved Solids.

COPPER (Cu). The element copper is found fairly often in trace amounts in natural water. Examinations by Kehoe[196] proved its presence in natural surface waters in quantities up to 0.068 ppm and in natural ground waters up to 12 ppm. Braidech [177] reported it in amounts varying from 0.005 ppm to 0.6 ppm after examining samples of domestic water supplies in 24 cities in the United States.

Copper may be accreted by domestic water by solution of copper and brass water pipes and other copper-bearing equipment in contact with the water. According to Hale,[224] a committee of the New England Water Works Association said that "* * * it has been shown that with acid water having a pH value of about 6.5, the copper content is generally only about 0.5 ppm even after standing in the pipe lines for an appreciable length of time." In the management of water purification systems, it is not uncommon practice to use copper sulfate in impounded bodies of water as an algicide, the normal dosage usually being less than 1.0 ppm.[255]

Copper is beneficial in human metabolism and a deficiency of it results in nutritional anemia in infants. It is estimated that the human adult has a daily turnover of 2.0 to 2.5 mg and that children of preschool age need 0.1 mg per kilogram of body weight daily for normal growth.[213] Excretion by the body in urine averages 1.0 mg per day; the remainder is eliminated in the feces. Normal diet plus that acquired from drinking water provides an adequate copper intake. Small amounts are not considered to be toxic but prolonged consumption of copper may result in damage to the liver.

Copper gives water an undesirable taste; the degree to which it is objectionable depends upon the taste perception of the consumer. At a concentration of 2.6 ppm in distilled water and 5.0 ppm in spring water, copper taste was apparent to 5 per cent of a test panel.[226] On the basis of taste, it is desirable to reduce the copper content of drinking water to below 1.0 ppm.[165]

IRON (Fe). Iron is one of the most abundant constituents of rocks and soils and will be found in many natural waters in bivalent or trivalent form. Industrial wastes and acid drainage from mines contribute iron to surface waters. Riddick [227] reports iron in certain North Carolina (U.S.) ground waters ranging from 2.0 to 5.0 ppm and Hem [171] states that in acid waters, iron may be present in amounts exceeding 100 ppm. Kehoe [196] found the mean concentration of iron in the drinking water supplies of 37 communities in the United States to be 0.3 ppm.

Iron is a very objectionable constituent in a finished drinking water, either for domestic or industrial use. It imparts a brownish color to laundered goods and stains plumbing fixtures, and it affects adversely the taste of beverages.

Water containing iron may taste bitter or astringent depending upon the amount of iron in the water and the acuity of the consumer's taste perception. Cohen et al [226] found that 5 per cent of a test panel could taste 0.04 ppm of ferrous iron in distilled water and 0.12 ppm in spring water.

According to Sollmann,[213] iron is essential to the metabolic activity of all mammalian cells. A normal American diet supplies, generally, from 11-19 mg of iron daily, an amount sufficient for nutrition. The average daily retention of iron by normal adults approximates 2.0 mg but as absorption is poor, larger quantities need to be taken. The iron that is not retained passes into the feces.

The recommended [165] limit beyond which the quantity of iron should not be permitted to rise in drinking water is 0.3 ppm. This limit is based upon the feasibility of its removal; this amount of iron has no toxicological significance.

MANGANESE (Mn). Manganese in natural water may be expected to be found in the bivalent and quadrivalent states. It is present there as a result of its solution from soils and sediment aided by bacteria or organic chemicals.

In most natural waters, the amount of manganese is less than 0.2 ppm. A higher concentration may be found in ground and surface waters but it does not ordinarily exceed 1.0 ppm [228] unless there are contributions from mining or other industrial operations. Sediments deposited in impounded waters may yield manganese from 1-10 years or longer after the impoundment has been filled. The amounts detected in such reservoirs have varied from 0.2-20 ppm.[229] In his study of the constituents in the drinking water of United States' cities, Kehoe [190] determined the mean concentration of manganese to be 0.017 ppm.

Manganese contaminates everything it touches, producing gray to black stains. It forms incrustations in water distribution pipes and reduces their carrying capacity, and it causes discoloration of industrial products. Like iron, manganese degrades the taste of drinks. Cohen and his associates reported it to be detectable by 5 per cent of a test panel when 3.4 ppm were present in distilled water and, by estimation, to 50 per cent when 180 ppm were concentrated in spring water.[226]

In mammals, manganese is an essential food element; a deficiency of it interferes with growth, blood and bone formation and reproduction, according to Sollmann.[213] Daily intake for man is about 10 mg; orally administered manganese passes chiefly into the feces, only a small amount being absorbed. Oral administration of it in non-dust form to animals results in hepatic cirrohosis. Prolonged exposure to excessive amounts of manganese in dust form leads to the principal toxic effect, namely, manganese toxicosis, with prominent cerebral symptoms. Kawamura [230] reported 16 cases of manganese poisoning, five of which were so severe that two of them died. These patients had consumed manganese-containing water of a well around

which 300 dry cells had been buried. The amount of manganese in the water was not given. There are no data to show the level at which ingested manganese becomes harmful to man.

Because of aesthetic and economic considerations, it is recommended [165] that the amount of manganese in drinking water be less than 0.05 ppm.

NITRATE (NO_3). Nitrogen may occur in water in several forms, depending upon the level of oxidation but nitrate, the completely oxidized state of nitrogen, is the principal form in most natural water. The nitrate content of surface waters free of pollution is seldom as high as 5.0 ppm and often less than 1.0 ppm. In ground water, however, the concentration may run from 0 to nearly 1,000 ppm.[171] The character of the plant life covering the ground, the use of nitrogen-bearing fertilizers for soil improvement, the discharge of sewage and other wastes into rivers and streams, and other factors affect the nitrate content of water.

In 1945, Comly [231] first implicated nitrates as the causative factor producing two cases of cyanosis with serious blood changes in infants in Iowa (U.S.). In one case, well water used in food preparation contained 140 ppm of nitrate and 0.4 ppm of nitrite ion; in the other case, the well water used had in it 90 ppm of nitrate and 1.3 ppm of nitrite ion. These and other cases of methemoglobinemia made evident the danger of high-nitrate water to the health of infants. Campbell's [232] discussion of a similar case in Northern Ireland indicated the presence of nitrate-contaminated water in other parts of the world. Donahoe [233] pointed out that breast-fed infants of mothers drinking high-nitrate water may be poisoned and that cows consuming it may give milk containing sufficient nitrate to produce an adverse effect.

In 1951, Walton [234] summarized the methemoglobinemia situation in the United States by pointing out that there had been more than 278 cases and 39 deaths reported. With respect to 214 of these cases, the available data indicated that 37.8 per cent were associated with infant food containing well water having a nitrate concentration in the range of 51-100 ppm; 43.1 per cent with a concentration of over 100 ppm; and the remainder with a concentration of 11 to 50 ppm. Evidence indicates that surface waters do not present a problem of this kind except in very unusual cases.

Sodium nitrate has been fed to rats in quantities up to 10 per cent of their diet for their lifetime and, other than some depression in growth at feeding levels above 1 per cent, no adverse effects were noted in them. When two dogs were given 2 per cent sodium nitrate in their food for 105 and 125 days respectively, there were no obvious adverse effects or disturbances in the blood.[235]

It has been hypothesized that the susceptibility of infants to methemoglobinemia is connected with the acidity of the gastric juices which act upon ingested nitrates. "When the acidity of the gastric juice is low, nitrate-reducing bacteria thrive in the upper intestinal tract, there reducing nitrates to nitrites, which in turn, are absorbed into the blood stream" causing a change in the hemoglobin of the blood.[225] Because the gastric juices of infants under six months of age are less acid than those of adults,

they are more susceptible; adults are not affected like infants by drinking the same nitrate-contaminated water.

In light of the many uncertainties attached to the presence of nitrate in drinking water, a limit of concentration beyond which its presence should not be tolerated is set at 45 ppm.[165] There is currently no single and economical method for removing excess nitrate from water. Therefore, in areas where it is known to be present beyond the above limit, the public should be warned of the potential danger of using high-nitrate water for infant feeding.

PHENOLS. The "term" phenols includes cresols and xylenols. These compounds may be present in raw waters used as sources of supply for water purification systems as a result of pollution by industrial wastes. Kremer and Cunjak[236] describe finding a concentration of 0.019 ppm in the lower Allegheny River (U.S.), 0.054 ppm in the lower Monongahela River (U.S.) and 0.07 ppm in wells adjacent to these rivers.

The amounts present would be of little significance were it not for the objectionable tastes and odors resulting when phenol-bearing water is disinfected with chlorine, a water purification practice now used extensively throughout the world. Baylis has estimated that the smallest amount of phenol that can be tasted in chlorinated water is considerably less than 0.01 ppm[237] and Cunliffe[238] states that the addition of a fraction of 1.0 ppm of elemental chlorine to phenol-contaminated water makes the phenol taste detectable in dilutions of 0.0025 to 0.005 ppm.

Heller[239] fed drinking water containing various dilutions of phenol to rats to study its action on them. He found no results unfavorable to these animals when phenol was present in the water in amounts ranging from 15 to 1,000 ppm. At 7,000 ppm the rats' growth was retarded and when the concentration rose to 10,000 ppm both growth and reproduction were hindered. A large amount of phenol is rapidly conjugated to less toxic substances and eliminated in the urine as rapidly as it is absorbed; considerable seems to be metabolized or lost.

Because of the very general custom of disinfecting drinking water with chlorine and the ability of the latter to intensify the repulsive taste of an extremely low concentration of phenolic compounds, 0.001 ppm is recommended as the upper limit to the presence of this substance in drinking water.[165]

SULFATE (SO_4). See Total Dissolved Solids.

TOTAL DISSOLVED SOLIDS. It is well known that many people regularly drink water, without any obvious ill effects, which contains amounts of chlorides, sulfates or total dissolved solids in excess of limits deemed acceptable by the modern water purveyor. More than 100 public water supplies in the United States contain over 2,000 ppm of dissolved solids. Some people adjust themselves to mineralized water and accept it without violent reaction but others do not and are likely to reject it, seeking a supply more to their taste. To a visitor or a new resident, a mineralized water undoubtedly has an intolerable taste.

Richter[240] used a panel of normal people to study their taste response

to sodium chloride in distilled water. He found that the panelists were able to distinguish a sodium chloride solution from distilled water in an average concentration of 160 ppm and that the salt taste was first recognized when the average concentration was 870 ppm. As long ago as 1907, Whipple[241] studied the taste-perceptiveness of about 20 people, using various salts in distilled water. The concentration at which each salt could be detected by the participants varied within the following lower and upper limits:

Salt	Range in ppm
Calcium chloride	150 — 350
Magnesium chloride	200 — 750
Potassium chloride	350 — 600
Sodium chloride	200 — 450
Calcium sulfate	250 — 900
Magnesium sulfate	400 — 600
Sodium sulfate	250 — 550

Lockhart,[242] using 18 or more persons as a panel, determined threshold concentrations in distilled water with the following conclusions:

Salt	Threshold Concentration in ppm
Calcium chloride	347
Potassium chloride	650
Sodium chloride	345
Magnesium sulfate	500

Water high in dissolved solids, particularly sulfate, is likely to exert a laxative effect on a consumer unaccustomed to it. Moore[243] summarized survey data available from users of water coming from 248 wells and with a dissolved solids content of over 1,000 ppm, most of which was sulfate in a majority of the wells. His conclusions were based upon positive statements with respect to the laxative effects. Water containing 1,000-2,000 ppm gave difficulty to 21 per cent of the survey respondents; above 2,000 ppm, more than 50 per cent of the people suffered a laxative result; and a concentration of 4,000 ppm was almost certain to be laxative. A concentration of sulfate and magnesium together is critical at about 1,000 ppm and when the concentration is higher than that, 60 per cent or more of the users will have trouble. More than 2,000 ppm of sulfate alone can be expected to produce discernible physiological effects.

Coffee brewed with highly mineralized water is less acceptable in taste than when made with water containing less chloride and sulfate.

The factors impelling the setting of limits of concentration for chlorides, sulfates and total dissolved solids in drinking water are generally aesthetic and economic ones. A gross chloride concentration makes water completely unusable for drinking and sulfates may be laxative which is annoying to some and intolerable to others but small amounts of these constituents can be and are borne by consumers if they do not move to acquire a supply with a better taste.

It is desirable in the production of a high-quality drinking water to hold the concentrations of the following constituents below the levels shown: chloride—250 ppm; sulfate—250 ppm; total dissolved solids—500 ppm.[165]

ZINC (Zn). Normally zinc would not be anticipated in natural ground and surface waters in more than trace amounts although Hem[171] has reported two mine waters containing 200 ppm and 345 ppm respectively. Braidech's[177] spectographic findings on the drinking water of American cities showed a zinc content ranging from 0.005 to 0.2 ppm and Kehoe[196] found a mean concentration of 0.136 ppm in the drinking water of 37 large communities in the United States.

Zinc is a beneficial element in human metabolism. The usual daily intake is 10-15 mg. Excretion of zinc is by way of the alimentary tract and very little is retained; feces contain about 10 mg and urine 0.4 mg each day.

The oral toxicity of zinc compounds is low and 30-40 ppm in drinking water is considered safe according to Sollmann.[213] Anderson[244] mentions several communities in which water containing zinc in amounts varying from 4.9 to 18.46 ppm was ingested without ill effects. Scott[245] has described the sole and non-harmful use of a drinking water with a fluctuating zinc content of 20.9-40.76 ppm for more than two years by 200 men at a military post.

Drinker[246] and her associates fed daily doses of 175 to 1,000 mg of zinc oxide to dogs and cats for 3 to 53 weeks. The animals' health was not affected; neither did histological examination following autopsy disclose any damage to their organs attributable to zinc.

Zinc salts act as gastrointestinal irritants; the ingestion of them in food where their taste is disguised produces an acute though transitory illness within a few minutes after ingestion. Rats deficient in zinc show a nutritional disturbance and their growth is only about one-third of that of normal ones.[247 213]

A metallic taste is imparted to water by 40 ppm of zinc.[244] Five per cent of a test panel detected zinc sulfate in distilled water when the concentration was 4.3 ppm.[226] If water containing an appreciable amount of carbon dioxide remains in contact with galvanized pipes or containers, zinc will almost surely be dissolved from them. Water takes on a milky appearance when it contains 30 ppm of zinc carbonate.[248]

Although zinc does not have poisonous qualities to seriously impair man's health when present in his drinking water, it is responsible for aesthetic effects which should be avoided; hence, zinc-contaminated drinking water should be accepted only when the zinc content is less than 5.0 ppm.[165]

COMPARISON OF THE INTERNATIONAL[249] AND THE U.S. PUBLIC HEALTH SERVICE STANDARDS FOR THE CHEMICAL CHARACTERISTICS OF DRINKING WATER *

Even though water may be free of living organisms and hence safe to drink, it may be unacceptable because it contains offending chemical or physical substances. Every water technician, in both urban and rural areas, has encountered situations in which drinking water has been obnoxious to its users because of chemical or physical shortcomings. Acceptability, pro-

* All figures are in milligrams per liter.

tection of health and conservation of the water system all require a drinking water of good chemical and physical quality.

The chemical composition of water in different parts of the world varies widely. Standards of quality covering chemical substances have been suggested by the World Health Organization [249] and these are shown below compared with similar standards now in use in the United States.

	International	Public Health Service
Arsenic (As)	0.2	0.05[a]
Barium (Ba)		1.0
Cadmium (Cd)		0.01
Chromium (hexavalent Cr)	0.05	0.05
Cyanide (Cn)	0.01	0.2[a]
Fluoride (F)	If present in excess of 1.0-1.5, dental fluorosis or worse effects may result. Optimum amount to prevent dental caries—1.0.	See table 9
Lead (Pb)	0.1	0.05
Selenium (Se)	0.05	0.01
Silver (Ag)		0.05
Nitrate (NO_3)	In infants under 1 year, 50-100 may give rise to infantile methemoglobinemia.	45

Where nitrate content is known to be higher than specified limit, public should be warned of potential danger of using water for infant feeding.

	Permissible[b]	Excessive[c]	Not to be Exceeded[d]
Alkyl Benzene Sulfonate (ABS)			0.5
Calcium (Ca)	75	200	.....
Carbon Chloroform Extract (CCE)			0.2
Chloride (Cl)	200	600	250
Copper (Cu)	1.0	1.5	1.0
Iron (Fe)	0.3	1.0	0.3
Magnesium (Mg)	50	150	.....
Magnesium plus Sodium Sulfate	500	1,000	.....
Manganese (Mn)	0.1	0.5	0.05
Phenols	0.001	0.002	0.001
Sulfate (SO_4)	200	400	250
Total Dissolved Solids	500	1,500	500
Zinc (Zn)	5.0	15	5.0

[a]—Where other more suitable water supplies are available at reasonable costs, these limits should be reduced to: arsenic—0.01; cyanide—0.01.

[b]—"The limits * * * designated 'permissible' apply to a water that would be generally acceptable by consumers." [249]

[c]—"Values greater than those listed 'excessive' would markedly impair the potability of the water." [249]

[d]—The substances listed should not occur in excess of the limits given where other more suitable water supplies are, or can be made, available at reasonable cost.[165]

REFERENCES

165. Public Health Service Drinking Water Standards-1962. Federal Register, 27:2152. Government Printing Office, Washington (1962).

166. Ohio River Pollution Survey—Industrial Waste Guides. Mimeographed papers. U.S. Public Health Service, Cincinnati, Ohio (1942).

167. Pollution-Caused Fish Kills. Multilithed paper. U.S. Public Health Service, Washington (1960).

168. Davidson, F. F. Poisoning of Wild and Domestic Animals by a Toxic Waterbloom of *Nostoc rivulare* Kuetz. Jour. AWWA, 51:1277 (1959).

169. Olson, T. A. Toxic Plankton. Water & Sewage Wks., 99:75 (1952).

170. Tisdale, E. S. Epidemic of Intestinal Disorders in Charleston, W. Va., Occurring Simultaneously with Unprecedented Water Supply Conditions. Am. J. Pub. Health, 21:198 (1931).

171. Hem, J. D. Study and Interpretation of the Chemical Characteristics of Natural Water. Geological Survey Water-Supply Paper 1473. Government Printing Office, Washington (1959).

172. Neal, P. A. et al. A Study of the Effect of Lead Arsenate Exposure on Orchardists and Consumers of Sprayed Fruit. U.S. Public Health Service Pub. Health Bull. No. 267 (1941).

173. Summary of Tolerances and Exemptions for Residues of Pesticide Chemicals. 2nd edition. Mimeographed paper. U.S. Food and Drug Administration, Washington (1957).

174. Hill, A. B. and Fanning, E. L. Studies in the Incidence of Cancer in a Factory Handling Inorganic Compounds of Arsenic: Mortality Experience in the Factory. Brit. J. Indust. Med., 5:1 (1948).

175. Doll, R. Occupational Lung Cancer: A Review. Brit. J. Indust. Med., 16:181 (1959).

176. Piper, A. M., Garrett, A. A. and others. Native and Contaminated Ground Waters in the Long Beach—Santa Ana Area, California. Geological Survey Water-Supply Paper 1136. Government Printing Office, Washington (1953).

177. Braidech, M. M. and Emery, F. H. The Spectrographic Determination of Minor Chemical Constituents in Various Water Supplies in the United States. Jour. AWWA, 27:557 (1935).

178. Threshold Limit Values for 1958. A.M.A. Arch. Indust. Health, 18:178 (1958).

179. Stokinger, H. E. and Woodward, R. L. Toxicologic Methods for Establishing Drinking Water Standards. Jour. AWWA, 50:515 (1958).

180. Frant, S. and Kleeman, I. Cadmium "Food Poisoning". J.A.M.A., 117:86 (1941).

181. Cangelosi, J. T. Acute Cadmium Metal Poisoning. U.S. Nav. Med. Bull., 39:408 (1941).

182. Lieber, M. and Welsch, W. F. Contamination of Ground Water by Cadmium. Jour. AWWA, 46:541 (1954).

183. Decker, L. E., Byerrum, R. U., Decker, C. F., Hoppert, C. A. and Langham, R. F. Chronic Toxicity Studies—Cadmium Administered in Drinking Water to Rats. A.M.A. Arch. Indust. Health, 18:228 (1958).

184. Ginn, J. T. and Volker, J. F. Effect of Cadmium and Fluorine on the Rat Dentition. Proc. Soc. Exper. Biol. & Med., 57:189 (1944).

185. Jacobs, E. E., Jacob, M., Sanadi, D. R. and Bradley, L. B. Uncoupling of Oxidative Phosphorylation by Cadmium Ion. J. Biol. Chem., 223:147 (1956).

186. Schroeder, H. A. Trace Metals and Chronic Diseases. Advances in Internal Medicine—Vol. VIII. The Year Book Publishers, Chicago (1956).

References (continued)

187. Graham, D. W. Chromium—A Water and Sewage Problem. Jour. AWWA, 35:159 (1943).

188. Davids, H. W. and Lieber, M. Underground Water Contamination by Chromium Wastes. Water & Sewage Wks., 98:528 (1951).

189. Machle, W. and Gregorius, F. Cancer of the Respiratory System in the U.S. Chromate-producing Industry. Pub. Health Rep., 63:1114 (1948).

190. Muehlberger, C. W. Possible Hazards from Chemical Contamination in Water Supplies. Jour. AWWA, 42:1027 (1950).

191. MacKenzie, R. D., Byerrum, R. U., Decker, C. F., Hoppert, C. A. and Langham, R. F. Chronic Toxicity Studies-Hexavalent and Trivalent Chromium Administered in Drinking Water to Rats. A.M.A. Arch. Indust. Health, 18:232 (1958).

192. Gross, W. G. and Heller, V. G. Chromates in Animal Nutrition. J. Indust. Hyg. & Tox., 28:52 (1946).

193. Smith, O. M. (Abstract) The Detection of Poisons in Public Water Supplies. Wtr. Wks. Eng., 97:1293 (1944).

194. Bodansky, M. and Levy, M. D. Studies on the Detoxification of Cyanids. Arch. Int. Med., 31:373 (1923).

195. Karsten, A. (Abstract) Effects of Cyanide on Black Hills Trout. Jour. AWWA, 28:660 (1936).

196. Kehoe, R. A., Cholak, J. and Largent, E. J. The Concentration of Certain Trace Metals in Drinking Water. Jour. AWWA, 36:637 (1944).

197. Dean, H. T. Endemic Fluorosis and Its Relation to Dental Caries. Pub. Health Rep., 53:1443 (1938).

198. Hodge, H. C. and Smith, F. A. Fluoridation as a Public Health Measure—Some Public Health Aspects of Water Fluoridation. Amer. Assoc. for the Advance. of Sci., Washington (1954).

199. Dean, H. T., Arnold, F. A. and Elvove, E. Domestic Water and Dental Caries. Pub. Health Rep., 57:1155 (1942).

200. Galagan, D. J., Vermillion, J. R., Nevitt, G. A., Stadt, Z. M. and Dart, R. E. Climate and Fluid Intake. Pub. Health Rep., 72:484 (1957).

201. Arnold, F. A., Dean, H. T., Jay, P. and Knutson, J. W. Effect of Fluoridated Public Water Supplies on Dental Caries Prevalence. Pub. Health Rep., 71:652 (1956).

202. Maier, F. J. Partial Defluoridation of Water. Pub. Wks., 91:90 (1960).

203. Howard, D. C. Lead in Drinking Water. Am. J. Pub. Health, 13:207 (1923).

204. Wright, W., Sappington, C. O. and Rantoul, E. Lead Poisoning from Lead Piped Water Supplies. J. Indust. Hyg., 10:234 (1928).

205. Negus, S. S. The Physiological Aspects of Mineral Salts in Public Water Supplies. Jour. AWWA, 30:242 (1938).

206. Kehoe, R. A., Cholak, J., Hubbard, D. M., Bambach, K., McNary, R. R. and Story, R. V. Experimental Studies on the Ingestion of Lead Compounds. J. Indust. Hyg. & Tox., 22:381 (1940).

207. Kehoe, R. A. Exposure to Lead. Occup. Med., 3:156 (1947).

208. Drill, V. A. Pharmacology in Medicine. McGraw-Hill Book Co., New York (2nd ed., 1958).

209. Dudley, H. C. Toxicology of Selenium. Am. J. Hyg., 23:169 (1936).

210. Fitzhugh, O. G., Nelson, A. A. and Bliss, C. I. The Chronic Oral Toxicity of Selenium. J. Pharmacol. & Exper. Therap., 80:289 (1944).

211. Hadjimarkos, D. M. and Bonhorst, C. W. The Trace Element Selenium and Its Influence on Dental Caries Susceptibility. J. Pediat., 52:274 (1958).

212. Just, J. and Szniolis, A. Germicidal Properties of Silver in Water. Jour. AWWA, 28:429 (1936).

References (continued)

213. Sollmann, T. A Manual of Pharmacology. W. B. Saunders Co., Philadelphia (8th ed., 1957).

214. Hill, W. R. and Pillsbury, D. M Argyria. The Williams and Wilkins Co., Baltimore (1939).

215. Kehoe, R. A., Cholak, J. and Story, R. V. Manganese, Lead, Tin, Aluminum, Copper, and Silver in Normal Biological Material. J. Nutrition, 20:85 (1940).

216. Walton, G. Effects of Pollutants in Water Supplies—ABS Contamination. Jour. AWWA, 52:1354 (1960).

217. Schwartz, A. M., Perry, J. W. and Berch, J. Surface Active Agents and Detergents, Vol. II. Interscience Publishers, Inc., New York (1958).

218. Deluty, J. Synthetic Detergents in Well Water. Pub. Health Rep., 75:75 (1960).

219. The Relation of Surface Activity to the Safety of Surfactants in Foods. Food Protection Committee, National Research Council, Pub. 463, Washington (1956).

220. Fitzhugh, O. G. and Nelson, A. A. Chronic Oral Toxicities of Surface-Active Agents. J. Am. Pharm. Assoc.-Sci. Ed., 37:29 (1948).

221. Freeman, S., Burrill, M. W., Li, T. W. and Ivy, A. C. Enzyme Inhibitory Action of Alkyl Aryl Sulfonate and Studies on Its Toxicity When Ingested by Rats, Dogs and Humans. Gastroenterology, 4:332 (1945).

222. Hollis, M. D. The Water Pollution Image. J. Wtr. Pol. Cont. Fed., 33:215 (1961).

223. Taylor, F. B. Incomplete and unpublished data obtained by the Division of Environmental Engineering and Food Protection, Public Health Service, Washington (1961).

224. Hale, F. E. Relation of Copper and Brass Pipe to Health. Wtr. Wks. Eng., 95:84 (1942).

225. Water Quality and Treatment. American Water Works Association, Inc., New York (2nd ed., 1951).

226. Cohen, J. M., Kamphake, L. J., Harris, E. K. and Woodward, R. L. Taste Threshold Concentrations of Metals in Drinking Water. Jour. AWWA, 52:660 (1960).

227. Riddick, T. M., Lindsay, N. L. and Tomassi, A. Iron and Manganese in Water Supplies. Jour. AWWA, 50:688 (1958).

228. Water Quality Criteria. State Water Pollution Control Board, Pub. No. 3, Sacramento, California (1952).

229. Griffin, A. E. Problems Caused by Manganese in Water Supplies. Jour AWWA, 50:1386 (1958).

230. Kawamura, R. and others. (Abstract) The Group Intoxication of Mangan by Drinking Well Water. Biol. Abst., 15;1119 (1941).

231. Comly, H. H. Cyanosis in Infants Caused by Nitrates in Well Water. J.A.M.A., 129:112 (1945).

232. Campbell, W. A. B. Methaemoglobinaemia Due to Nitrates in Well Water. Brit. Med. J., II, 371 (1952).

233. Donahoe, W. E. Cyanosis in Infants with Nitrates in Drinking Water as Cause. Pediatrics, 3:308 (1949).

234. Walton, G. Survey of Literature Relating to Infant Methemoglobinemia Due to Nitrate-Contaminated Water. Am. J. Pub. Health, 41:986 (1951).

235. Association of Food and Drug Officials of the United States. Quarterly Bull., 22:136 (1958).

236. Kremer, R. W. and Cunjak, R. J. Problems and Progress in Reducing Phenolic Tastes. Jour. AWWA, 45:495 (1953).

References (continued)

237. Baylis, J. R. An Improved Method for Phenol Determinations. Jour. AWWA, 19:597 (1928).

238. Cunliffe, R. W. Chlorophenol Tastes in Milwaukee's Water Supply. Jour. AWWA, 14:423 (1926).

239. Heller, V. G. and Pursell, L. Phenol-contaminated Waters and Their Physiological Action. J. Pharmacol. & Exper. Therap., 63:99 (1938).

240. Richter, C. P. and MacLean, A. Salt Taste Threshold of Humans. Am. J. Physiol., 126:1 (1939).

241. Whipple, G. C. The Value of Pure Water. John Wiley and Sons, New York (1907).

242. Lockhart, E. E., Tucker, C. L. and Merritt, M. C. The Effect of Water Impurities on the Flavor of Brewed Coffee. Food Research, 20:598 (1955).

243. Moore, E. W. Physiological Effects of the Consumption of Saline Drinking Water. Committee on Sanitary Engineering and Environment, National Research Council, Washington (1952).

244. Anderson, E. A., Reinhard, C. E. and Hammel, W. D. The Corrosion of Zinc in Various Waters. Jour. AWWA, 26:49 (1934).

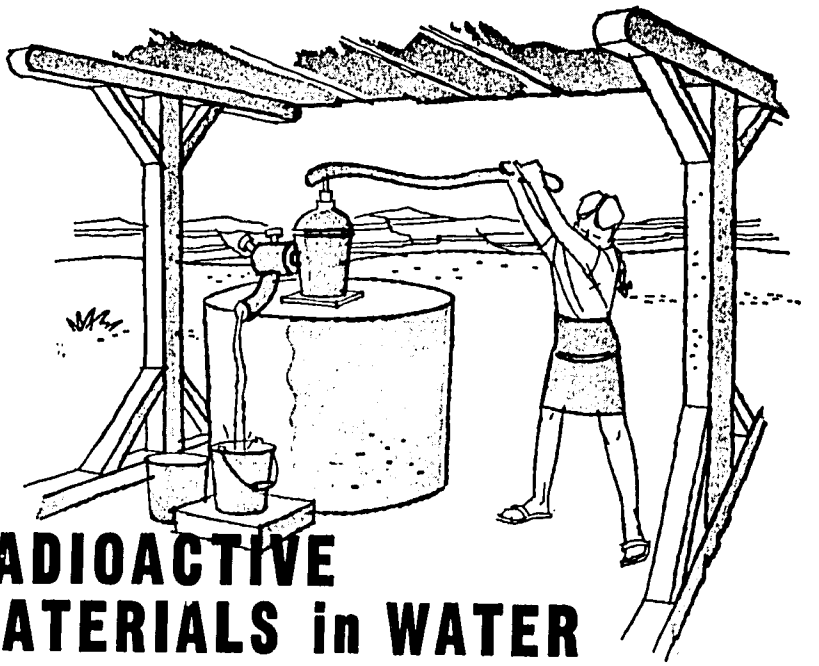
245. Scott, H. H. and Jameson, W. W. Zinc Waters—Their Potability and Germicidal Effect. Lancet, I:1008 (1917).

246. Drinker, K. R., Thompson, P. K. and Marsh, M. An Investigation of the Effect of Long-Continued Ingestion of Zinc, in the Form of Zinc Oxide, by Cats and Dogs, Together with Observations upon the Excretion and the Storage of Zinc. Am. J. Physiol., 80:31 (1927).

247. Vallee, B. L. Zinc and Its Biological Significance. A.M.A. Arch. Indust. Health, 16:147 (1957).

248. Drinker, C. K. and Fairhall, L. T. Zinc in Relation to General and Industrial Hygiene. Pub. Health Rep., 48:955 (1933).

249. International Standards for Drinking Water. World Health Organization, Geneva (1958).



RADIOACTIVE MATERIALS in WATER

Man is continuously exposed to minute amounts of ionizing radiation * coming from his environment but this natural exposure is not sufficient to result in any discernible physiological effect. With the development of the nuclear industry and the utilization of its products, some small amount of additional radiation exposure is unavoidable.

Any human radiation exposure should be looked upon as harmful and any unnecessary exposure should be avoided. Ionizing radiation injures man by producing a harmful effect on the body's living cells; bone marrow may be damaged by chronic exposure to low concentrations of radium^{226} and strontium ^{90}, two of the most hazardous radionuclides.

Avoidance of radiation exposure is decidedly the best way to escape injury but today complete isolation from man-made radiation is not always possible in some parts of the world. If it is assumed that some artificial radiation will be present in man's environment, action should be directed to minimizing it to the greatest extent feasible.

* The safe limits of concentration set forth here are drawn from the U.S. Public Health Service Drinking Water Standards. (165)

As some wastes from the nuclear industry often reach water courses, the presence of radioactive materials in drinking water drawn from them becomes a matter of concern. Currently the nuclides radium ^{226} and strontium ^{90} are of such importance that they merit particular attention in any consideration of the quality and safety of the drinking water used by man.

SAMPLING PERIOD. When the concentration of radium ^{226} or strontium ^{90} in drinking water varies considerably, test samples should be composited over a period of three months. With respect to gross beta concentration, test samples should be taken more frequently.

RADIUM ^{226} and STRONTIUM.^{90} Drinking water containing radium ^{226} and strontium ^{90} is acceptable without further question when the concentration does not exceed 3 uuc * and 10 uuc per liter, respectively. If the water supply is known to be the only source of radioactivity, these levels can be increased provided the total body intake does not exceed 20 uuc and 200 uuc per day, respectively. Where other radionuclides are present in the environment, the permissible levels of concentration of radioactivity in the water should be such that when added to the concentrations from other sources, the total daily body intake will not exceed 20 uuc and 200 uuc, respectively.

GROSS BETA ACTIVITY. In the absence ** of strontium ^{90} and alpha emitters, a water suitable for drinking should not contain a gross beta concentration exceeding 1,000 uuc per liter except when more complete analyses indicate that concentrations of nuclides are not likely to cause exposures greater than those established by the U. S. Federal Radiation Council.[250 251]

> "Look to your health; and if you have it, praise God, and value it next to a good conscience; for health is the second blessing that we mortals are capable of; a blessing that money cannot buy."[252]

REFERENCES

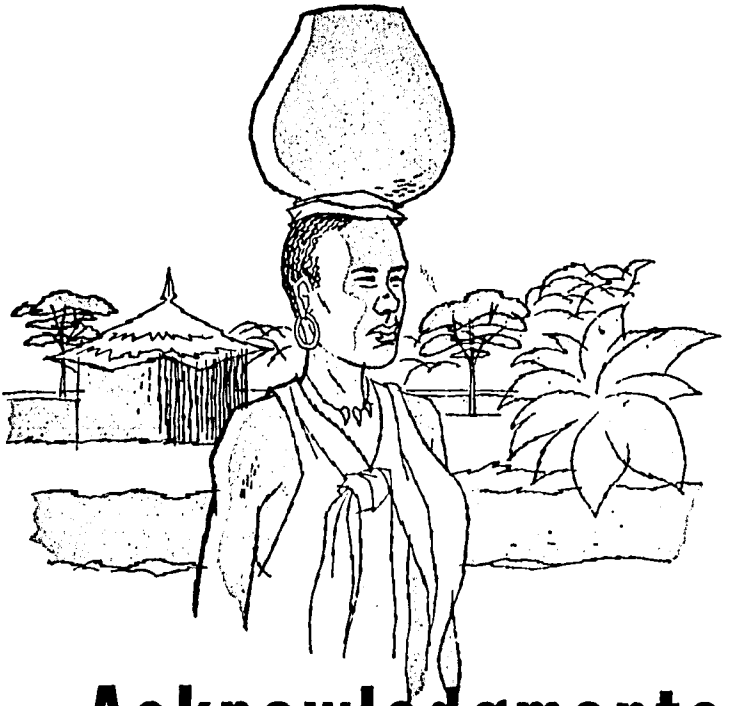
250. Radiation Protection Guidance for Federal Agencies. Federal Radiation Council. Federal Register, 26:9057. Government Printing Office, Washington (1961).

251. Backround Material for the Development of Radiation Protection Standards. Federal Radiation Council, Report No. 2. Government Printing Office, Washington (1961).

252. Walton, Izaak. Compleat Angler, Chap. 21.

* uuc = micromicrocurie

** Absence is interpreted to mean a negligibly small part of the amounts in the limits established for radium ^{226} and strontium ^{90}.



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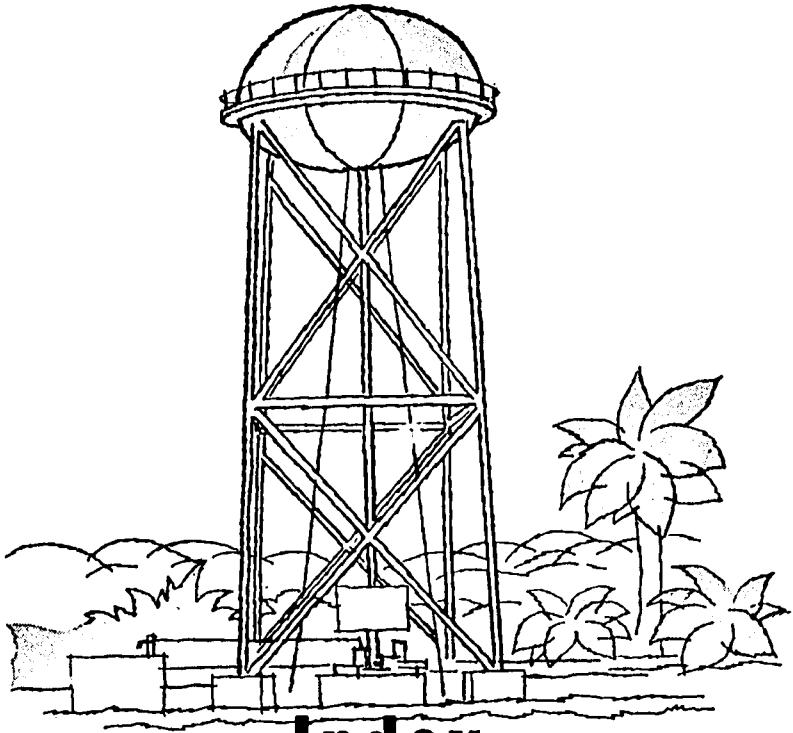

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